THE
BRITISH JOURNAL OF SURGERY

VOL. XV. APRIL, 1928. No. 60.

EPOCH-MAKING BOOKS IN BRITISH SURGERY.

BY SIR D'ARCY POWER, K.B.E., LONDON.

IV.

THE WHOLE COURSE OF CHIRURGERIE COMPILED BY PETER LOWE, SCOTCHMAN.

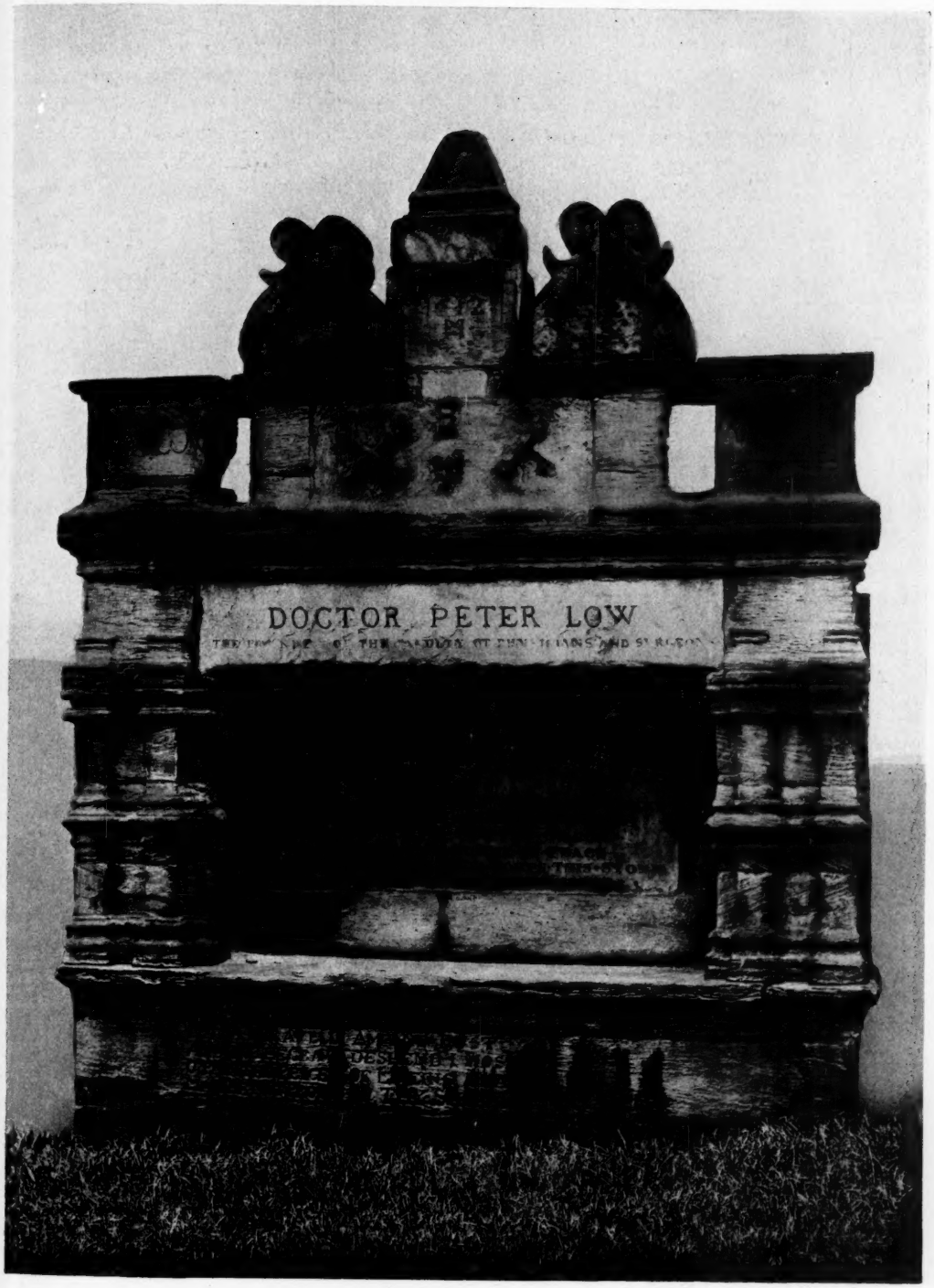
PETER LOWE describes himself as Doctor in the Faculty of Surgery in Paris and Surgeon in Ordinary to the most victorious and Christian King of France and Navarre. The first edition of the book was published in 1597, the second in 1612, both being printed by Thomas Purfoot during the lifetime of Lowe; the third and fourth editions appeared posthumously. The first edition is a small book without illustrations; the second, third, and fourth were greatly enlarged, and have woodcuts copied from Guillemeau and Ambroise Paré.

The book differs from those of Gale and Clowes because it is written for the use of students who were going to be examined in surgery, whilst Lowe's predecessors had written for those who were already in practice. It was compiled, as Master Peter Lowe says, in the second edition, for the use of his son John, "because I am mindful to cause you to remain in Paris for your further instruction and passing of the degrees there accustomed in the said Art [of surgery] for the which I will in these three books following, as also in every general chapter set up as briefly as I can the form and method that the Doctors of Chirurgerie in Paris useth in their first examination—called the examination tentative—where the Provost of the said College and six other Masters that he will choose will examine you exactly upon the whole ground of Chirurgerie." John, however, never entered medicine. Lowe had already published a small work on the Spanish Disease, and he intended to write a treatise on the sickness of women; another on Infantment and a Poor Man's Guide. None of these works can now be traced, so that it is probable they were never issued, perhaps never written. "The whole Course of Surgery" is partly a catechism, the interlocutors being John Cointret, Dean of the Faculty of Surgery in Paris, and Peter Lowe his scholar in the first edition, Peter Lowe

and his son John in the second and subsequent editions; partly a series of more elaborated chapters, some of which give details of his own experiences. We learn from the book that most of his surgery was the result of military service, that it was not very extensive, and that he was somewhat timid. He was an honest, straightforward person who had as hearty a hatred of quacks and unlicensed practitioners as Gale and Clowes. Thus, in answer to the question, "What method is to be observed of the Chirurgian in working his operations?" the answer is, "First to know the disease, next to do the operation as soon as may be, surely, without false promises or deceit to heal things that cannot be healed, for there are some, who void of knowledge and skill promise for lucre to heal infirmities being ignorant both of the disease and the remedies thereof. These faults be often committed of some who, usurping the name of Chirurgian, being unworthy thereof, have scarce the skill to cut a beard which properly pertaineth to their trade."

There are many interesting chapters, and not the least is that dealing with the tonsils. It is headed, "Of the Tumor of the Amigdalles, called by the Greeks Parischimia and by Latines Tonsillæ", in which the operation of tracheotomy is described. After detailing the symptoms and constitutional treatment he says: "If it aposthume [suppurates], as often happeneth, it must be opened with a lancet, if the tumor doth so augment that the sick is in danger to suffocke then thou shalt make incision on the trachearter, betwixt the third and fourth ring, cutting only the membrane that holdeth them together and not the proper substance of the cartilage, so the air shall avoid [escape], put in a tent of silver that is hollow into it till the inflammation be past, thereafter close up the wound. This was practised by Andrew Scot one of the King of Scotland's Chirurgions in Paris most cunningly. To do this operation thou shalt hold backward the patient's head, lift up the skin with the hand and cut it in length, so the membrane shall be discovered, then make your incision with a bistourie in the said place; during which time, use externally, medicaments about the neck that are made of barley flour, lint seed and fenegree as shall be found most expedient according to the degree of the malady. This operation of incision is very dangerous if it be not well made by reason of the flux of blood which often doth happen as also for the nerves which pass that way. I cured an apothecary in Paris after this method and he healed."

Speaking of pleurisy he says: "If by these remedies the pain doth not cease, neither that ye perceive any evacuation of the humour, neither by the mouth, urine, nor fundament it is to be suspected to turn into Empiem which is a collection of matter between the ribs and the region of the lights, sometime with corruption hereof, for the which we make incision either by cautery or lancet but rather by the cautery, for neither doth it close so soon, nor is so dolorous. The opening shall be between the third and fourth of the true ribs, beginning at the nethermost and so counting upward, six inches from the ridge of the back. If ye see tumour eminent in any place of the thorax, open it in the most convenient place. When it is open put in a hollow tent either of silver or lead. Let not all the matter avoid at one time, but little by little as other apostumes." Ten years later he adds in the second edition: "This cure is rare and seldom practised, yet after this method I did cure John



PETER LOWE'S TOMB.

Buchan, Maister of the Song School in Glasgow, who was heavily grieved with a watery or congealed humor collected under the sternum."

As regards tumours in the scrotum he recognized encysted hydrocele of the cord, for he says: "The signs are, the tumour is clear and becometh long, still in one estate, not painful heavy, sometime hard and is known by holding the cod between thee and the candle and being enclosed in membranes it appeareth to be a third testicle. As for the cure you must make incision in the side of the Codde, eschewing the testicle, put a tent in it and dress it twice a day, keep it open till the humor be evacuated, use remedies anodious [anodynes] for to appease the dolor and cicatrise it as other wounds."

Lowe was greatly in advance of his time in describing an operation for the relief of strangulated hernia. The operation for the cure of reducible inguinal hernia was often done, but even two hundred years later the unfortunate patient with a strangulated hernia was allowed to die unrelieved. Speaking of an intestinal rupture Lowe says: "The signs are these;—there cometh inflammation and the more ye press to reduce it, the inflammation is the greater, so it changeth the colour which is an evil sign, swooning and voyding of matter at the mouth are evil signs, and if the intestine be not reduced the patient dieth which happeneth through the narrowness of the dilatation. As for the cure, first rub the codd with oil of camomile and lay the sick on his back in such sort that his arse be higher than his head, reducing with thy hand little by little, pressing most on the place where it descended. If the fæcal matter let [prevent] the reduction use the remedies set down in Bubonocele and clysters to discharge the intestine; if by these remedies the intestine do not reduce but the matter wax hard with great dolor, ye shall make incision in the upper part of the cod, eschewing the intestine, thereafter put a little piece of wood up by the prolongation of the peritoneum near the hole of descent, the piece of wood must be round on one side and flat on the other, whereon ye shall make the rest of your incision and rub the descent with a little oil, so it shall easily reduce. If then it reduce not, the peritoneum must be incised and use the cautery Gastrographick and handle it as other wounds. This operation must not be used but in great necessity and when the sick is strong, prognosticating still of the danger ne fefellisse aut ignorasse videaris." (It is clear that he was possessed of the native Scottish caution.) "Being reduced it must with bandages and astringent fomentations bee retained."

Lowe did not hesitate to recommend operations for the cure of varicocele and of varicose veins. "The signs of varicocele are, the repletion of the veins, sometime few in number, sometime many wrapped together like a vine branch, soft in touch and returning into the belly by pressing on the didyme. The judgments [signs] are it is without dolor, most dangerous and difficil to be cured. The cure shall be first purging the body of melancholick, then bleed if need be, thereafter make incision on the scrotton the breadth of two fingers in the place of the varix, thereafter make pass a needle with double thread under the varix one in the upper part of the wound, another in the lower part, leaving an inch betwixt, then open the varix and evacuate the humor contained; if there be any moe do the like, that done knit the thread and handle the wound as others."

The chapter on varicose veins is equally good and practical. It is headed, "Of the tumor in the veins of the legs called Varicæ". "Varix is a dilatation of the vein greater than natural, which happeneth in divers parts of the body, as temples, belly, under the navel, on the testicles and matrix but chiefly in the legs, which is sometime one vein, sometime divers together, full of thick, burnt, melancholick blood letting [preventing] action of the place. The cause is abundance of the melancholick humor, vehement exercise as leaping, wrestling, carrying of great burthens, strokes, falls, torments and such like. The signs may be seen by the greatness of the veins, which commonly are blacker than the natural; The judgements, those that are intern are incurable and not to be touched. The cure is diversely set down by divers authors, some counsel to incise them in divers places according to the circumvolution and let them blood, then to close up and bind the wound as in other bloodings, resting that day and if they swell again do the place with a little ink the skin above the vein, thereafter take up the skin with thy hands and incise in the midst thereof; then let it go, that the vein may be seen and pass two needles as in the varicose hernia. Let it bleed a quantity and knit it. Let the thread fall off of itself without drawing it by force and cure the wound as others."

We are indebted to Mr. Annan, of Glasgow, for the photograph of the monument of Maister Peter Lowe in St. Mungo's churchyard.

PERITONEAL LYMPHATIC ABSORPTION:

AN EXPERIMENTAL INVESTIGATION TO DETERMINE THE VALUE OF LYMPHATICOSTOMY.*

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In 1922 Costain[1] first introduced his procedure of drainage of the thoracic duct—lymphaticostomy—as a means of treating severe cases of acute peritonitis. It was based on a series of experiments carried out on dogs suffering from experimentally produced peritonitis. The primary lesion was set up by ligating the appendix and meso-appendix, and it was found that this produced what was claimed to be a lethal peritonitis. In a series of 7 dogs the appendix and meso-appendix were ligatured and the thoracic duct was drained; 3 of these dogs recovered. In a further series of 11 dogs with peritonitis the thoracic duct was ligated; in 4 of these dogs recovery ensued, and in each of them a thoracic duct fistula had developed. Believing that the escape of toxic material from the thoracic duct was the essential factor in the recovery of these animals, Costain applied his procedure to patients suffering from acute generalized peritonitis, and in his paper he quoted the case of one patient who recovered.

At first sight this method of treatment is attractive, and it has been adopted by a number of surgeons with very varying results. Similarly experimental results have varied greatly. Cox and Bell,[2] Lewin and Copher,[3] and McGuire[4] have all carried out experiments very similar to those of Costain, but have failed to obtain his results. McGuire injected cultures of *Bacillus prodigiosus* into the peritoneal cavity of dogs, and in only one case was a positive culture obtained from the thoracic duct. Williamson and Brown,[5] using the same organism, obtained a culture from the duct in 4 out of 8 animals.

The end-results in cases of acute peritonitis are very variable. Patients almost *in extremis* will make a rapid recovery after the primary lesion is dealt with and the peritoneal cavity drained, while others, apparently less seriously ill, fail to respond to operative treatment. It is thus impossible at the present time from a small series of cases to assess the true value of lymphaticostomy on a purely clinical basis.

In the light of this conflicting evidence, both clinical and experimental, it seemed possible that a study of the lymphatic drainage of the peritoneal cavity with particular reference to the passage of peritoneal effusions along the thoracic duct might throw some light on the value or otherwise of this new technique.

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EXPERIMENTAL RESULTS.

The experiments upon which this paper is based were carried out in rabbits, cats, and dogs. The fluid injected was 2 per cent colloidal silver in saline, a solution which had been used previously by Bolton[6] in similar work. This solution is particularly suitable as it is only absorbed by the lymphatics. It stains the lymphatics and glands brown or black according to the length of time it has been in contact with the tissues. All the experiments were carried out under anæsthesia. In the case of rabbits, urethane was used chiefly, but in some cases intratracheal ether was administered. The larger animals were always anæsthetized with ether. At the end of the experiment the animals were killed with chloroform.

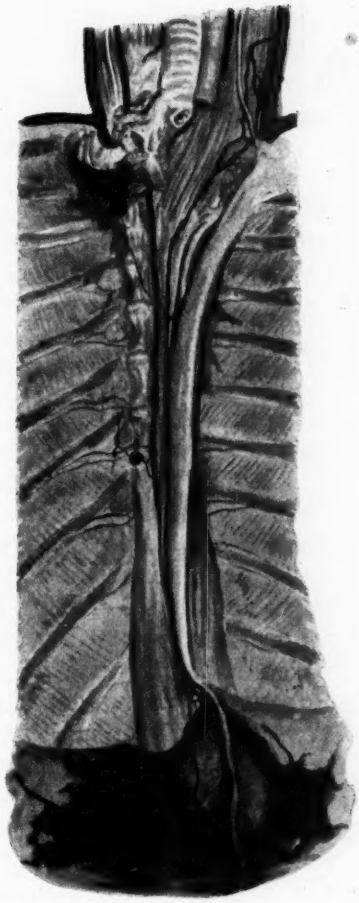

FIG. 383.—Rabbit. The lymphatics are seen passing from the diaphragm into the thoracic duct, which is deeply stained and can be traced up to the neck. The branching of the duct in the neck is more complex than is usually seen. Note the small stained gland to the right of the spine. The upper drawing shows the appearance at the root of the neck on the left side two minutes after injection.

Group 1.—Injections of 2 per cent colloidal silver into the peritoneal cavity of rabbits. The animals were killed at periods varying from ten minutes to two hours after injection. The amounts injected varied from 40 c.c. to 10 c.c., but the results obtained were the same, regardless of the amount of fluid injected.

FINDINGS.—The findings in this series were as follows:—

Diaphragm.—The staining was very uniform. The central area was almost black; immediately to the right of this the staining was less dense but still well marked, while to the left it was much less evident. This was so, even when the injection was made under the left cupola of the diaphragm. Passing from the diaphragm three or four main lymphatic vessels could be traced to their junction with the thoracic duct. One of these vessels came from the left side and the remainder from the right (*Fig.* 383). At the extreme lateral margins of the diaphragm a well-stained lymphatic vessel could

always be traced forwards to its junction with the internal mammary lymphatics.

Internal Mammary Lymphatics.—These lymphatics, passing up on the deep aspect of the sternum, were stained by the colloidal silver within ten minutes of the intraperitoneal injection. When traced upwards they were found to drain into a number of glands situated in the region of the first intercostal space. In many cases lymphatics could be seen passing from these glands to the right and left cervical veins, quite distinct from the thoracic duct.

Thoracic Duct.—The duct was always deeply stained and could be traced up to its junction with the left cervical vein. The greater part of the silver appeared to reach the duct from the diaphragm, as that portion of the duct lying between the receptaculum chyli and the junction of the three diaphragmatic lymphatics was less deeply stained than the part above. Passing from the duct as it lay on the posterior thoracic wall were several small lymphatic vessels which drained into lymphoid nodules scattered irregularly on each side of the vertebral column. The receptaculum chyli became stained much more slowly than the duct, and the silver appeared to reach it by way of the retroperitoneal lymphatics.

If the thoracic duct was ligated previous to the intraperitoneal injection, the duct became distended with colloidal silver up to the point of ligature, while the internal mammary lymphatics were much more deeply stained than usual. These lymphatics evidently took on the function of the ligated duct so far as absorption from the peritoneal cavity was concerned.

Group 2.—Injections of colloidal silver into the peritoneal cavity of rabbits, a thoracic duct fistula having previously been made.

FINDINGS.—Within two minutes of the injection the silver was discharging from the fistula in the neck, and continued to do so until the animal was killed.

In this series of experiments an attempt was made to determine the essential factor in promoting the flow of chyle through the thoracic duct. Though the results were inconclusive they may be referred to briefly.

Costain states that there is a suction force on the thoracic duct owing to the venous blood passing its opening. This is evidently of little importance; otherwise complete division of the thoracic duct would tend to stop the flow, which is the reverse of what is found both clinically and experimentally. Respiratory movement is also considered to be responsible for the flow along the duct. "With each respiration there occurs a temporary negative pressure within the thorax which draws the chyle from the abdominal cavity." To determine the true effect of respiration, colloidal silver was injected into a rabbit, the thorax having been laid widely open previously under intratracheal anæsthesia. The whole of the thoracic wall on both sides was removed, only the sternum and the attachments of the diaphragm being left intact. Under these conditions it was possible to observe the duct throughout its thoracic course, and it was found that there was no delay in the appearance of the dye; within thirty seconds of its injection the lower part of the duct was stained by the silver and it rapidly discharged through the fistula in the neck.

From these findings it would appear that there is some propulsive action forcing the chyle up the duct, and it may be that the diaphragm plays an important part.

In several rabbits, with the duct divided in the neck and the thorax widely opened, both phrenic nerves were divided. This procedure caused a definite delay in the appearance of the silver, but within ten minutes the duct was well stained. This experiment does not, however, rule out diaphragmatic action, as the phrenics are not wholly responsible for the supply of the diaphragm and their division does not stop all diaphragmatic movements.

Group 3.—Intraperitoneal injections of colloidal silver in cats and dogs.

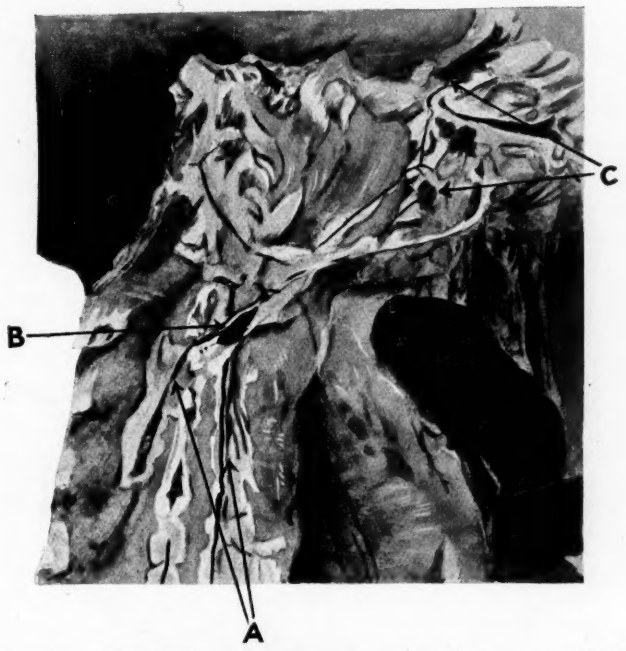

FIG. 384.—Cat. The sternum has been turned aside to expose its deep aspect. Note: A, The internal mammary lymphatics; B, The gland; C. The efferent lymphatics passing from the gland to the veins at the root of the neck. All these structures are stained by the colloidal silver.

FINDINGS.—The peritoneal lymphatic absorption in these animals was found to be totally different from that occurring in rabbits. No staining of the thoracic duct took place until two hours had elapsed, and then only faint traces of the dye could be seen at its lower end. The diaphragm showed definite staining within thirty minutes of injection; but this was never well marked, and lymph-vessels were never seen passing back to join the duct. The diaphragmatic lymphatics drained directly to the internal mammary lymphatics, which were always well stained within half an hour of injection. As in the rabbit, these lymphatics drained to lymph glands on the deep aspect of the sternum and from there efferent vessels could frequently be traced to veins at the root of the neck. These vessels had a very variable distribution;

they opened into the blood-stream directly and not by the thoracic duct (*Fig.* 384).

In several cats colloidal silver was injected directly into the bowel wall. Following this the dye could be seen streaming down the mesenteric lymphatics, and within a few minutes it appeared in the thoracic duct, previously exposed in the neck. On killing the animal the mesenteric lymphatics were traced to glands situated on the upper part of the posterior abdominal wall, and from them lymphatics passed to the receptaculum chyli

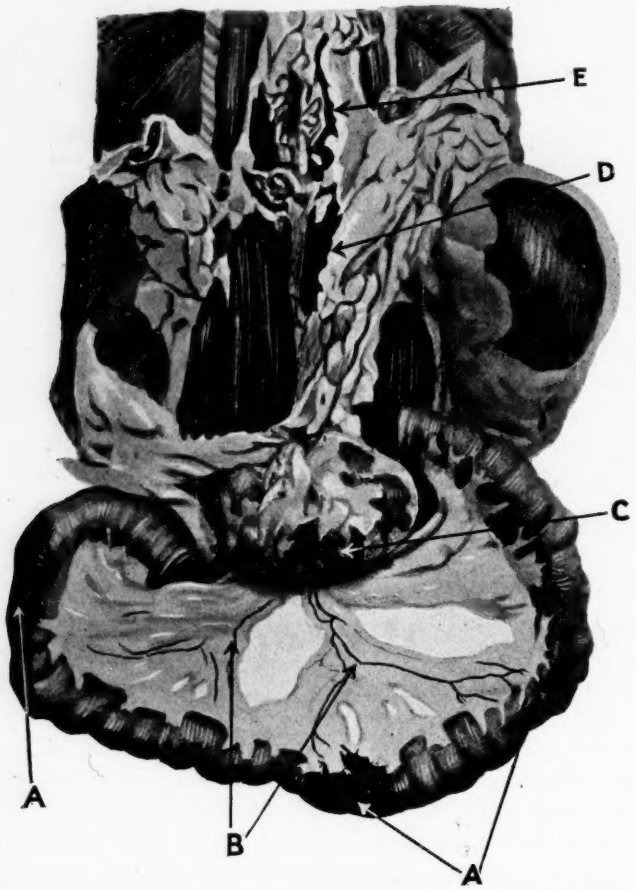

FIG. 385.—Cat. At three points (A) injections have been made into the bowel wall. Note: B, The lymphatics passing down the mesentery to a gland (C) on the posterior abdominal wall. This gland was abnormally enlarged previous to the injections, but it shows definite staining. The receptaculum chyli (D) and the thoracic duct (E) are distended with colloidal silver.

(*Fig.* 385). In none of these experiments were the internal mammary lymphatics stained.

Group 4.—Post-mortem findings in a dog dying of acute generalized peritonitis.

FINDINGS.—The abdomen contained a large quantity of purulent fluid which had spread throughout the peritoneal cavity. Examination of the

thorax showed that the glands on the deep aspect of the sternum, into which the internal mammary lymphatics drained, were very markedly enlarged and congested (*Fig.* 386), while the thoracic duct and posterior mediastinal glands showed no abnormality.

Group 5.—Removal of the omentum and observation of the rate of absorption via the thoracic duct in rabbits.

Findings.—If the animal was killed fifteen minutes after injection, the diaphragm was only slightly stained, only the lower part of the duct contained colloidal silver, and the internal mammary lymphatics were unstained. If the animal was allowed to live for longer periods, the staining was present as in *Group* 2 but was not so well marked.

As removal of the omentum usually resulted in some hæmorrhage into the peritoneal cavity, it was thought that this might cause delay in absorption, and therefore in another series of rabbits a vessel was allowed to bleed into the peritoneal cavity and the colloidal silver was then injected. It was found that this produced delay exactly similar to that noted when the omentum was excised.

Injections made directly into the omentum never resulted in staining of the duct or other lymphatics, and it would appear that it plays little part in the lymphatic absorption from the peritoneal cavity.

Fig. 386.—Sternum of dog, showing numerous enlarged and congested lymphatic glands lying on the deep aspect of the upper part of the sternum. There were no pathological changes in the posterior part of the thorax.

Group 6.—Effects of alteration in position on lymphatic absorption.

Dandy and Rowntree[7] have shown that absorption by the blood-stream varies in different areas of the abdomen. They find that 15 per cent less of the dye injected is absorbed from the pelvis than from the upper abdomen.

When rabbits were put in the Fowler position and silver was injected, it was found that there was practically no alteration in the rate of absorption, owing to the fact that the fluid passed very rapidly to the upper abdomen. If, however, special precautions were taken to ensure that the silver was injected directly into the pelvis and the animal was kept in the vertical position, there was a definite delay in the appearance of the colloidal silver in the thoracic duct. At the end of one and a half hours, staining was well marked up to the level of the kidneys, and there was a minute trace under the diaphragm. The diaphragm, thoracic duct, and internal mammary lymphatics were unstained. On the posterior part of the central portion of the diaphragm a small nodule, stained brown, was seen, and on section this was found to be a lymphoid nodule containing colloidal silver.

SUMMARY OF EXPERIMENTAL FINDINGS.

1. In rabbits, absorption takes place very rapidly by the lymphatics of the upper abdomen and especially by the diaphragm, although retroperitoneal lymphatics play a subsidiary part. The main path of absorption is by the thoracic duct.

2. In cats and dogs, absorption by the thoracic duct from the peritoneal cavity is a very slow and unimportant process. The function of the duct is to drain the chyle from the intestines. The internal mammary lymphatics are stained early and drain to the anterior mediastinal glands.

3. The omentum plays little, if any, part in the actual lymphatic absorption from the peritoneal cavity.

4. The paths of lymphatic absorption in cats and dogs are in certain respects similar to those of man.

This last statement requires proof, and in support of it may be quoted certain observations made by Durham[8]:—

In a study of 30 fatal cases of peritonitis in man he found the anterior mediastinal glands swollen, congested, and containing organisms. (Compare case of dog in *Group* 4.)

In one case of ruptured tubal abortion the same glands were found to be distended with blood.

Very seldom have organisms been found in the chyle from the thoracic duct.

In three fatal cases of peritonitis where the anterior mediastinal glands showed no changes the omentum was adherent to the anterior and lateral parietes, thus cutting off the diaphragm from the infected area.

CONCLUSIONS.

From all the above observations two facts would appear to emerge:—

1. The fact that no other observer has been able to obtain Costain's experimental results is explained, because there is little absorption by way of the thoracic duct in cats and dogs.

2. Lymphaticostomy cannot be recommended as a procedure holding out any hope to patients suffering from acute peritonitis.

I am much indebted to Professor D. P. D. Wilkie for his helpful advice and criticism, and to Mr. John Grieve for the illustrations.

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[2] Cox and Bell, *Ann. of Surg.*, 1925, lxxxi, 911.
[3] Lewin and Copher, *Arch. of Surg.*, 1925, x, 742.
[4] McGuire, *Surg. Gynecol. and Obst.*, 1925, xl, 626.
[5] Williamson and Brown, *Amer. Jour. Med. Sci.*, 1923, clxv, 480.
[6] Bolton, *Jour. Pathol. and Bacteriol.*, 1921, xxiv, 429.
[7] Dandy and Rowntree, *Beitr. z. klin. Chir.*, 1913, lxxxvii, 539.
[8] Durham, *Jour. Pathol. and Bacteriol.*, 1897, iv, 338.

RIGHT-SIDED VISCEROPTOSIS:

AN ESTIMATE OF THE IMPORTANCE OF ABNORMAL MOBILITY AND PROLAPSE OF THE ASCENDING COLON AND CÆCUM IN THE CAUSATION OF VARIOUS ABDOMINAL CONDITIONS, BASED ON OBSERVATIONS IN A SERIES OF 242 CASES TREATED BY RIGHT COLOPEXY.

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3. Abnormal Mobility and Prolapse of the Ascending Colon and Cæcum: (*a*) Varieties and Degrees; (*b*) Anatomical and Pathological Results.
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III.—Clinical Types and Secondary Results.

IV.—Relationship with Certain Forms of Appendicitis.

V.—Relationship with Gastric and Duodenal Ulceration and Gall-bladder Conditions.

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VII.—Anatomical and Pathological Results of Colopexy.

VIII.—Clinical Results of Colopexy.

IX.—Conclusions.

Being impressed, in 1920, by Waugh's[1] claims for the success of the treatment of right-sided ptosis by right colopexy, I decided to form a personal estimate of the value of this method. The investigation soon assumed broader lines, and included a study of abnormal intra-abdominal membranes and their relation to ptosis and stasis, and also an estimate of the importance of abnormal mobility and prolapse of the ascending colon and cæcum as a factor in the causation of various abdominal conditions. My endeavour throughout has been to base my conclusions not on theoretical grounds but on the anatomical, pathological, and more especially the clinical conditions and results.

For the whole-hearted co-operation of my assistants and various radiologists I am more than grateful. My thanks are also due to many 'family

doctors'—the ultimate judges—who, welcoming any form of treatment which would offer their most troublesome patients a reasonable chance of cure, have been only too willing to keep me informed of the progress of these patients after operation.

I. ANATOMICAL AND PATHOLOGICAL CONSIDERATIONS.

1. ABNORMAL INTRA-ABDOMINAL BANDS OR MEMBRANES: THEIR ORIGIN AND THEIR RELATION TO PTOSIS AND STASIS.

There are several well recognized, and not uncommon, bands or membranes found in definite localities of the abdomen about the origin and significance of which there has been great difference of opinion. Lane,[2, 3, 4, 5] to whom great credit is due for his pioneer work, is of the opinion that they are acquired and 'evolutionary' in origin, being 'a mechanical sequel to the assumption of the erect position'. He believes that a measure of stasis, due to erect posture and increased by faulty habits in childhood or by frequent pregnancies, results in delay in the fæcal current; that this is first noticed in the pelvic colon, and extends to the whole of the large bowel, with resulting dilatation and prolapse of the cæcum and the ascending and transverse colons. The result of this is, he holds, in the 'vigorous subject' the formation of bands. These are formed by the drag of the dilated prolapsed bowel on its mesenteric attachments, causing a 'peritoneal reaction', the result of what he calls "crystallization of the lines of strain." This is at first merely a streak or 'fine condensation' on the surface of the mesentery, but soon spreads to the bowel itself, and ultimately becomes a distinct membrane which is separated from the mesentery except at each end. These bands, which are thus considered to be due to the "effort of the organism to meet an abnormal loading up of the bowel because of a distal obstruction", may at first serve a beneficial purpose by supporting the loaded bowel and thus enabling it to "perform its function to the best of its capacity and not to prolapse". Later, however, they tend to cause obstruction by diminishing its lumen, partly by angulation and partly by axial rotation. He states, further, that this does not take place until the bands 'begin to contract', but at the same time says that " no one familiar with their appearance, structure, or mode of development could regard them as inflammatory".

The view that these bands are inflammatory in origin, originally suggested by Jackson,[6] and later supported by Morris,[7] Gerster,[8] Hertzler,[9] Hofmeister,[10] and Pilcher,[11] can be dismissed if only by the 'astonishingly regular anatomical character of the best examples'. (H. M. W. Gray and Anderson.[12]) In support of this one may also refer to the linear distribution of the vessels, which is quite unlike the radiating arrangement seen in inflammatory adhesions, and also to the fact that these thin membranes are not found along the line of the mesenteric lymphatics.

In refusing to accept the theory that they are congenital in origin, Lane quotes the results of Chapple's examination of 52 newly-born fœtus. Chapple could find no trace of "bands similar to those present in the adult". Against this must be brought the results of Eastman,[13] Flint,[14] Connell,[15] Pringle,[16] and Reid,[17] who show conclusively that the membranes are frequently present at birth.

H. M. W. Gray and Anderson give a much more reasonable and logical explanation of their origin by attributing them to excess, or in some cases failure, of developmental physiological fusion or absorption of membranes which exist in embryo. The fact that the symptoms connected with these membranes assert themselves usually for the first time in adult life cannot be used as an argument against their congenital origin any more than the late appearance of an inguinal hernia is evidence against the congenital origin of the sac. That the membranes often exist without any symptoms is rather in favour of than against the probability of a congenital origin. Tyrrell Gray,[18] also, referring to the gradual fixation of the colon in its evolution in man, suggests that these membranes represent variations in the process of mesenteric fusion and fixation in transitional types. In the detailed description

of the various membranes seen in my cases their developmental origin will be repeatedly substantiated, and it may, I think, be taken as established beyond doubt that they are congenital and developmental, not acquired or inflammatory.

Just as there has been much controversy regarding their origin, so has there been great diversity of opinion regarding their relation to the occurrence of ptosis and stasis. The two problems are interdependent.

As already stated, Lane holds that the occurrence of the membranes is secondary to stasis and ptosis; but this explanation cannot be accepted if the membranes are acknowledged to be congenital. Walton[19] considers that the membranes are merely variations in the normal development, and usually give rise to no symptoms. While not agreeing with this description of their character, H. M. W. Gray and Anderson admit that there are great variations in the effect produced. This may be due not merely to the variety of organs implicated, but also to the actual method of attachment of the membranes.

It is generally accepted that what may be described as a 'faulty body formation', although occasionally congenital, is much more commonly an acquired condition. Smith,[20] Goldthwait,[21] and Rovsing[22] have described two types, the 'virginal' and the 'maternal'. In the former the patient, who as a child was probably taller than normal, is thin and poorly developed; the thorax is narrow and shallow, with a small epigastric angle; the upper abdomen is also narrow and the waist long; the shoulders are round and the lumbar curve is diminished; the abdominal muscles are weak. These conditions are usually due to want of proper food and suitable exercise, and are found more commonly in girls than in boys. In the maternal type the thorax may be normally developed and the upper abdomen wide, but the abdominal muscles have been stretched by frequent pregnancies and the lower abdomen is prominent; the lumbar curve is lost, and in many cases there is loss of weight.

It is very difficult to say what constitutes an abnormally low position of the viscera. Bryant,[23] examining post mortem 290 subjects who had exhibited no symptoms of visceroptosis, found that in 10 per cent there was extreme visceroptosis and that in only 42 per cent was there none. As long ago as 1885, Treves,[24] examining 100 subjects, found that in 26 per cent there was some degree of mobility of the ascending colon due to persistence of its fœtal mesentery. No one, again, who is accustomed to seeing many opaque-meal examinations can doubt for a moment that some degree of prolapse and mobility of the ascending colon frequently occurs.

Many children are undoubtedly born with developmental membranes and a considerable degree of congenital ptosis who do not develop any symptoms of stasis. Thus we find many healthy adults with visceroptosis, people who are in good health although the stomach may reach below the interiliac line, and the cæcum hang well over the pelvic brim. Absence of symptoms in spite of this condition of ptosis is due to the fact that there has been no derangement of the mechanism of peristalsis. The viscera have retained their functional activity; the stomach, though low, is orthotonic; the cæcum is not dilated although in the pelvis.

This is analogous to the case of the child who may have a patent processus funicularis and yet not develop a hernia. The potential disability, in either case, need not necessarily develop into an actual disability provided that the other determining factors remain balanced, and that the general strength of the body is proportionate to the strain imposed on it. If, however, a weakness develops or an extra call is made, the balance may be disturbed. Thus, in the case of the processus funicularis, if the walls of the inguinal canal are weakened, or if there is an increase of intra-abdominal pressure, a hernia may develop. So, also, in the case under consideration. If the child with membranes and a degree of congenital ptosis due to failure of the normal process of fusion is poorly fed, and does not have suitable exercise, there is a loss of extraperitoneal fat and loss of the lumbar curve; the psoas shelf, on which the upper abdominal organs should be supported, is thus inadequate, and, in addition, the abdominal muscles, which should hold the viscera on the shelf, become weak (Coffey[25, 26]). The degree of ptosis is thus exaggerated, and the onset of stasis is accelerated if the ptosed organs are given more work to do—for example,

by indiscretions in diet or by carelessness in evacuation of the colon. If the membrane formation is slight, if the mesenteries are long, there may be very marked general visceroptosis with few symptoms; but if the viscera are held up at certain points by definite well-formed membranes, the effect of the ptosis will be much more marked. More will be said on this point when describing the clinical symptoms.

The onset of the symptoms, which synchronizes with the onset of stasis, is thus met with at puberty in the virginal type of faulty body formation, and more often in girls than in boys. In the maternal type the disturbance of the balance and the onset of symptoms usually occur soon after a pregnancy when the patient returns to active life.

A recent theory regarding the relation between these membranes and the occurrence of ptosis has been brought forward by Netschajew,[27] who, finding that the membranes contained longitudinal bundles of unstriped muscle fibres, suggests that if these are innervated by the sympathetic nerves, the ptosis caused by their relaxation may be the result, not the cause, of the nervous condition from which these patients so often suffer. The very difficult problem of the mental factor in visceroptosis will, however, be dealt with later.

The Lower Ileal or 'Lane's' Membrane.—About the frequent occurrence of this membrane, first described by Lane, there is no question, although his explanation of its origin is not accepted. Towards the end of fœtal life the lower end of the ileum enters the cæcum from behind, and it is easy to understand how the fusion of the posterior layer of the mesentery of the ascending colon with the dorsal parietal peritoneum, beginning at the hepatic flexure, may, by extending lower than usual, include the mesentery of the terminal ileum. If fusion of the peritoneum covering the terminal ileum with the dorsal parietal peritoneum takes place, it is drawn out, during the subsequent development of the ileum and cæcum, in the form of a triangular or quadrilateral membrane attached externally to the parietal peritoneum of the iliac fossa and internally to the antimesenteric border of a varying length of ileum. It is loosely connected to the posterior layer of the mesentery and the under surface of the ileum. I have seen, in one case, in addition to a well-marked Lane's membrane, evidence of excessive developmental fusion on the corresponding anterior surface of the mesentery.

In one of the cases in the series under review the terminal four inches of the ileum were lying entirely behind the dorsal peritoneum across the psoas muscle at the level of the iliac crest. The ascending colon was abnormally mobile and prolapsed, but the cæcum was held up in the iliac fossa by a very long appendix which was completely buried in 'developmental adhesions', its tip extending up almost to the liver. In this case fusion had evidently involved the appendix and the terminal ileum before descent, descent of the cæcum taking place later. Armstrong[28] reports two similar cases of "absence of a mesentery for the terminal six or seven inches of the ileum", in both of which the cæcum was mobile and in neither of which was there any sign of inflammation. He is of the opinion that this abnormal extension of the area of fusion is due to the drag on the mesentery of the terminal ileum by the 'genito-mesenteric fold', the superior border of which is attached to the under surface of the mesentery along the line of the iliac branch of the ileocolic artery.

A somewhat similar condition of the terminal ileum was seen in another of my cases, in which there had been a complete arrest in the descent of the cæcum. This, with the appendix, was firmly fixed to the posterior abdominal wall just below the liver, whereas the ascending colon, whose mesentery had persisted, had fallen down over it. The terminal three inches of the ileum were firmly held to the posterior abdominal wall by an unusually well-developed Lane's membrane. In several other cases in this series a varying length of terminal ileum was devoid of mesentery.

Lane's membrane is thus quite clearly caused by excessive irregular developmental fusion—either an abnormal extension downwards of the normal area of fusion, or an abnormally early commencement of fusion.

Too much importance has been attached to this membrane: however interesting it may be from the developmental point of view, it seldom causes any symptoms.

H. M. W. Gray and Anderson report that, although a Lane's membrane was found in 10 per cent of cases in which the right iliac region was examined, in 'only about half' of these could any symptoms be ascribed to it. In this series of 242 cases the membrane was present in 22·8 per cent, but as in all the cases there was irregularity in, or failure of the process of, fusion of the mesentery of the ascending colon, this greater frequency is not surprising. In very few could I convince myself that the membrane was responsible for any symptoms.

Lane, while adhering to his 'evolutionary' theory of the origin of the membrane, that its presence is the result not the cause of stasis, quite illogically states that its 'continued contraction' frequently causes obstruction of the lumen of the bowel. Now, although one cannot deny the possibility of kinking or axial rotation by this membrane, with or without abnormal mobility of the cæcum, even a slight degree of mechanical obstruction is very rarely seen. The possibility might perhaps not be fully appreciated, as the condition is seen with the patient in the supine position, were it not for the fact that hypertrophy of the proximal ileum, which would necessarily accompany any appreciable degree of mechanical obstruction, would be very evident.

Tyrrell Gray, discussing the possible association of ileal stasis with a Lane's membrane, does not accept the theory of mechanical obstruction. He states that he has never seen hypertrophy of the proximal ileum, but holds that the membrane, by causing increased tension in the mesentery, may result in stimulation of the sympathetic nerves distributed to the ileocæcal region with inhibition of the terminal ileum, spasm of the ileocæcal sphincter, and ileal stasis. I have no doubt that, by binding down the terminal ileum at a comparatively early stage in its development, the membrane may cause a very definite strain on the ileocolic vessels. This has been seen by me in two cases in which there was no prolapse of the ascending colon, and also in one case in this series; in all of them division of the membrane immediately relieved the strain on the vessels, the loop of ileum seeming almost to spring up into its normal position. The importance of this drag on the ileocolic vessels, or this increased tension in the mesentery, as a factor in the causation of chronic duodenal dilatation will be referred to later.

Peri-appendicular Membrane.—The cæcum, in its descent to the right iliac fossa, normally increases both in length and breadth; but the appendix, first seen as an appendage of the cæcum at the third month, does not show the same increase, and at birth is merely a narrow tube attached to the apex of the cæcum and not so sharply differentiated from it as in later life. This 'infantile' type of appendix may persist, and is often found associated with abnormal mobility and prolapse of the ascending colon and cæcum, the appendix being wide-mouthed and usually containing fæces. The cæcum may retain its infantile conical shape, with the infantile appendix hanging from its apex, but it usually increases in size and becomes quadrate by the development of two saccules, one on either side of the anterior longitudinal band. In the small percentage of cases in which the saccules develop equally, the appendicular attachment lies in the middle of the cæcum between the saccules. In the majority of cases, however, the outer or right saccule grows more rapidly and the point of attachment is pushed over to the left and may be in close proximity to the ileocæcal junction. It is, of course, more or less behind the cæcum.

During the descent of the cæcum the appendix may become implicated in the fusion and absorption of the mesentery of the ascending colon. As has already been noted when describing Lane's membrane, this implication may take place either in its final position in the iliac fossa or in any part of its descent. The appendix may thus come to be entirely retroperitoneal, lying behind the ascending colon and extending up as far as the liver, to which it may be 'adherent'. Or again, it may be merely attached, at the tip or elsewhere, to the posterior abdominal wall, or to the inferior surface of the mesentery of the terminal ileum. Peri-appendicular membranes of varying extent were present in 21 per cent of the cases in this series.

The difficulty of differentiating between these developmental membranes and true inflammatory adhesions which have arisen independently or in consequence of previously existing developmental membranes, will be referred to later when criticizing different methods of treatment.

If the appendix is fixed, in whole or in part, by such developmental membranes to the posterior abdominal wall or iliac fossa, it may act as a 'suspensory ligament' holding the cæcum, which in these cases is almost always abnormally mobile, out of the pelvis. This is the condition described by Lane as the 'controlling appendix', but his explanation that "in consequence of the strain exerted by the loaded cæcum the appendix becomes attached by acquired adhesions" cannot be accepted. The possible importance of this condition, which was seen in 10 per cent of my cases, in inducing inhibitory stimulation in the ileum and cæcum will be more fully dealt with later.

Mesosigmoid Membranes.—Membranes varying greatly in extent are frequently seen in the lower part of the left iliac fossa on the outer side of the mesosigmoid. Lane's theory that this 'first and last kink', as he has described it, is 'evolutionary' and acquired, the result not the cause of stasis, cannot be accepted. The membranes are undoubtedly developmental in origin, being due to irregular excessive fusion, "an attempt on the part of the mesosigmoid to become adherent, in the same way as does the ascending and descending mesocolon, to the dorsal parietal peritoneum" (H. M. W. Gray and Anderson[29]). I cannot agree with Lane's estimate of their enormous importance; although frequently seen in this series of cases, in very few did they require surgical interference. Stretching as they do from the mesosigmoid to the left iliac fossa, they might, if extensive or substantial enough, cause angulation or axial rotation of the bowel with consequent stasis. The theory that any pathological effect they may have is not mechanical but inhibitory in character will be more fully dealt with later.

Mesocolic-jejunal Membranes.—Mayo[30] has described a membrane stretching from the under surface of the mesocolon, just above the point of emergence of the small bowel, to the antimesenteric surface of the first part of the jejunum, which may be patchy or take the form of a definite triangular membrane, the length of jejunum involved varying considerably. There can be no doubt that this membrane is due to the drawing out of irregular excessive developmental fusions occurring in the mesocolon when that mesentery, after rotation of the colon, took up its new position and formed a new line of attachment crossing the duodenum.

In several of my cases I have seen this membrane, and have also noted other variations—for example, from the mesocolon to the anterior surface of the third part of the duodenum, across the duodenojejunal flexure, etc.

These membranes must not be confused with the *musculus suspensorius duodeni*, described by Treitz, a small flat muscle with a large proportion of fibrous tissue which, arising from the left crus of the diaphragm and passing downwards to the superior border of the duodenojejunal flexure, is continuous with the longitudinal and circular bands of the bowel.

The effect of these membranes depends on the points of attachment. There may be angulation of the jejunum a short distance from its origin, or there may be constriction of the third part of the duodenum; but these possible causes of duodenal stasis must be distinguished from the drag on the superior mesenteric vessels.

Right and Left Intercolic Membranes.—The downward extension, from the third month of fœtal life, of the bursa omentalis across the transverse colon to form the great omentum, may overlap and involve the upper portions of the ascending and descending colons.

Various observers have drawn attention to bands or membranes across the hepatic flexure. Pilcher has described the 'adhesion' of the ascending colon in its whole length to the transverse colon; Tyrrell Gray refers to a "membranous attachment of the cæcum and ascending colon to the proximal third of the transverse colon"; and Kuprijanoff,[31] writes of "strands and inflammatory or embryological adhesions" drawing the proximal and distal parts of the flexure together. A very definite extension of the great omentum across the hepatic flexure was seen in 37·5 per cent of my cases. In a few it was confined to the hepatic flexure alone; in the majority it extended at least half way down the ascending colon; and in a few it reached as far as the cæcum. In a few cases it extended across the bowel to be attached to the lateral parietes, but in all it was fused to and inseparable from the

anterior aspect of the ascending colon at the anterior longitudinal band. This 'membrane' is quite definitely part of the great omentum, exhibiting the same characteristics—for example, free anastomosis of blood-vessels, deposition of fat between its layers—and is certainly not inflammatory in origin. Confirmation of this view was obtained in a case of non-rotation of the colon. The cæcum lay in the left side of the pelvis, and although the colon passed in an almost straight line up to a very highly-placed splenic flexure, one could see where the hepatic flexure would have been had rotation taken place, by the presence of a slight angulation caused by such an extension of the great omentum.

I have never seen this extension of the great omentum across the hepatic flexure without some degree of abnormal mobility of the flexure, and the reason why the hepatic is involved much more frequently than the splenic flexure is undoubtedly because of the much greater frequency with which the former is abnormally mobile.

Acute angulation at the splenic flexure, with adhesion of the adjacent surfaces of the transverse and descending colons, described by Payr,[32] was attributed by him to traction on the 'phrenocolic ligament' with secondary inflammatory adhesions. The phrenocolic ligament is the name given to the peritoneal fold continuous with the left border of the great omentum, extending from the splenic flexure to the under surface of the diaphragm, and in cases of highly-placed splenic flexure it may appear short and tense, and in addition there may be true paracolonic inflammatory adhesions. I agree, however, with H. M. W. Gray and Anderson that Payr places too much importance on this, and am convinced that angulation at the splenic, as at the hepatic flexure, is more often due to an omental extension. This 'left intercolic membrane' was seen in four of the cases in this series, in one being accompanied by a 'right intercolic membrane'; in all four the splenic flexure was abnormally low and mobile; in only one did the membrane extend to the lateral parietal wall.

This omental extension, whether across the hepatic or splenic flexure may, by causing increased angulation, aggravate stasis in the proximal segment of the colon.

Jackson's Membrane.—This membrane was described by Jackson as "a thin vascular veil in which long, straight, unbranching vessels course, most of which are parallel with each other, and take a slightly spiral direction over the colon from the outer upper peritoneal attachment to the inner lower portion of the gut, ending just above the caput". I fail to understand Waugh's[33] statement that he had "for nine years sought in vain for Jackson's membrane", as the frequency of its occurrence is universally acknowledged. In this series of cases it was seen in 32 per cent. It varied greatly in extent. In most of the cases it was confined solely to the middle third of the ascending colon; in some the cæcum, and in a few the hepatic flexure, was also involved; in several cases the cæcum alone was involved. The membrane reaches as far as the anterior longitudinal band but no farther. It can be lifted up from the outer side and front of the bowel, where its attachment is very loose, but it cannot be separated from the anterior longitudinal band. As Jackson points out, "the colon seems placed in a diaphanous bag slightly too short to contain it without wrinkling", and on being handled "it slips about in its bag without entire freedom, as a fœtus within its amniotic sac".

I cannot agree with Jackson that the membrane holds the bowel 'in rigid plication', nor with H. M. W. Gray and Anderson that owing to its irregular distribution "the colon or cæcum may be tucked up in folds by its bands". The characteristic sacculation of the large bowel is due to the fact that the longitudinal bands are almost one-half shorter than the other coats of the bowel wall. Again, I cannot agree with Jackson that at the hepatic flexure the "drawn membrane particularly angulates the contained colon". Nor do I believe with H. M. W. Gray and Anderson that "it may produce very acute angulation at the hepatic flexure, where it is usually most marked"; nor that if spread over the appendix, as it occasionally is, it will, "by preventing free movement and evacuation of its contents, predispose to attacks of inflammation of that organ". The membrane is so thin and filamentous, it is so loosely attached to the bowel and can so easily adjust itself to the varying length of the colon in its descent, that I find it impossible to believe that it can have any obstructive effect or limit the mobility of the bowel.

That this membrane is developmental in origin cannot be doubted, but I do not accept H. M. W. Gray's[34] explanation of its formation. He affirms that "the chief cause of non-descent of the cæcum appears to be abnormal entanglement thereof by the right margin of the omentum . . . if the cæcum ultimately overcomes this obstruction to its descent, the affected part of the omentum becomes drawn out into Jackson's membrane . . . a well-marked Jackson's membrane can always be shown to be directly continuous with the ordinary omentum." Walton[35] also is of the opinion that a Jackson's membrane may cause fusion of the ascending and transverse colons, "in which case it is clearly a continuation of the great omentum". The transition I have never seen, nor can I understand the possibility of continuity between structures so absolutely unlike. In those cases in which I found present both a Jackson's membrane and a right intercolic membrane, the difference in their appearance and the absence of any communication, even when they actually overlapped, did not suggest a common origin. (*See Fig.* 387.) Again, in almost one-half of the cases in which there was a Jackson's membrane no right intercolic membrane was present. But an even more conclusive proof that Jackson's membrane does not originate from the great omentum in the way described by H. M. W. Gray is the fact that the descent of the cæcum, commencing as it does at the end of the third month of fœtal life, is well advanced before the great omentum has reached the lower border of the transverse colon. In a case of undescended cæcum I noted a Jackson's membrane involving the cæcum alone. In the only case in which I have seen "the counterpart of Jackson's membrane on the left side" it involved the whole of the descending colon, was well formed, and there was no suggestion of the bag being too short for its contents.

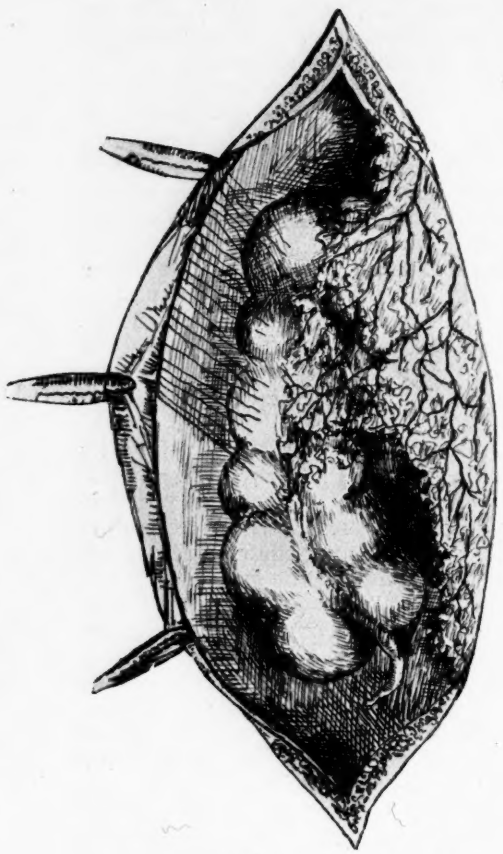


FIG. 387.—Showing a Jackson's membrane (with long, straight, parallel, unbranching vessels) and a right intercolic membrane (the extension of the omentum across the ascending colon).

It thus seems to me quite definite that this membrane originates at the end of the third month or the beginning of the fourth; that it descends with the bowel to which it is attached; and that the diaphanous bag is too short for the bowel it contains owing to the continued development of the latter after the formation of the former. The membrane is of no surgical importance.

Developmental Membranes involving the Liver, Gall-bladder, Duodenum, Hepatic Flexure, and Right Kidney.—The primitive ventral mesentery of the stomach and duodenum (the leaf of mesoblast which extends from the lesser curvature to the anterior abdominal wall) is, by the development of the liver, divided into three parts: the portion between the liver and the anterior abdominal wall and the diaphragm, the middle portion which forms a peritoneal covering for the liver, and the portion between the liver and the lesser curvature of the stomach and the duodenum. Of this last portion the part attached to the stomach persists as the lesser omentum, but the part attached to the duodenum normally disappears when the dorsal mesentery fuses with the posterior parietal peritoneum, and that

part of the gut becomes retroperitoneal and fixed. Irregularities in the disappearance of this part of the primitive mesentery, from the under surface of the liver to the duodenum, are common. The free right border of the lesser omentum is even less universally normal than the right border of the great omentum, and a great variety of developmental membranes may be found.

The most common abnormality is the persistence of the mesentery of the gall-bladder, which in its formation as an evagination from the common duct carried with it a fold of primitive mesentery. This may be merely a slight flimsy membrane at the apex of the gall-bladder, or the gall-bladder may have a complete mesentery extending from the fundus, or from the liver itself, down the cystic duct to the free right border of the small omentum. In 26 of my cases this condition was present in varying degrees. The membrane may, however, be continued as the 'hepatoduodenal' or 'cysticoduodenal' to the first and second parts of the duodenum. This was seen in 20 additional cases. In 14 other cases a 'hepatocolic' or 'cysticocolic' membrane stretched from the liver or gall-bladder to the gastrocolic omentum, the transverse colon, or hepatic flexure. In a few cases I have seen isolated membranes from the under surface of the liver to the duodenum (7 cases), from the pylorus and duodenum to the transverse colon (6 cases), and from the duodenum to the hepatic flexure (4 cases); in none of these was there a ventral membrane of the gall-bladder.

I cannot agree with H. M. W. Gray and Walton that these membranes may be actually continuous with the upper border of a Jackson's membrane. In one of my cases in which the upper attachment of a Jackson's membrane extended to the under surface of the liver external to the gall-bladder there was no difficulty in differentiating it from the cysticoduodenal membrane also present.

In one case a well-marked hepatocolic membrane contained within its layers, at a considerable distance from the liver, a nodule of liver tissue, thus proving conclusively that this membrane at least is of congenital origin. Short membranes are occasionally found connecting the hepatic flexure with the under surface of the right lobe of the liver; these are evidently the result of abnormal fusion between the two layers of peritoneum which should normally separate these structures. Such a condition was seen in 9 of my cases; in 2 the membranes were multiple, in 4 it was accompanied by, but entirely separate from a gall-bladder ventral membrane. The description 'hepatocolic' must be applied to these also, although their mode of development is different from those already so described.

In connection with the right kidney, also, developmental membranes are sometimes found resulting from either abnormal persistence or fusion. Although in those portions of the right kidney in contact with the duodenum and hepatic flexure the two peritoneal layers originally separating them normally disappear by absorption, these structures remaining attached to the kidney merely by loose areolar tissue, persistence may occur. The upper part of the 'genito-mesenteric fold', referred to when describing Lane's membrane, may thus persist as the 'duodenorenal ligament' of Huschke, and the hepatic flexure may be attached by a membrane, the 'colicorenal ligament', to the kidney. Again, short 'hepatorenal' membranes are occasionally found between the liver and the right kidney which are due to abnormal fusion between the two peritoneal layers normally separating these organs.

As the length and attachment of these different membranes vary so much, one need not be surprised at the variations in the effect produced by them—the effect depending also on the relative position, normal and abnormal, of the organs to which they are attached. This will be more fully dealt with when describing the anatomical and pathological results of abnormal mobility of the ascending colon and cæcum.

2. ANATOMICAL POSITION AND RELATIONS OF THE NORMAL ASCENDING COLON AND CÆCUM.

When considering the anatomical position and relations of the normal ascending colon and cæcum (*Fig.* 388), one must remember that, while the radiologist can visualize the bowel in the erect position, it is examined by both anatomist and surgeon in the supine position only. The cæcum is normally situated in the right iliac fossa, resting,

in the supine position, on the iliacus muscle and extending partly across the psoas muscle. Although usually entirely covered by peritoneum, the upper part of the posterior wall may not be so covered, being attached to the iliac fascia by loose areolar tissue. It has thus a considerable degree of freedom, and may extend, in the erect position, as far as the inner margin of the psoas muscle, thus reaching, but not passing over, the brim of the pelvis. Variations in its shape, due chiefly to variation in the development of its saccules, have already been referred to. The ascending colon, beginning at the ileocæcal valve as the direct continuation of the cæcum, passes upwards and slightly backwards, crossing the posterior part of the iliac crest, with its posterior surface connected by loose areolar tissue to the anterior surface of the quadratus lumborum muscle. Turning slightly forwards it reaches, or actually crosses, the anterior surface of the lower outer part of the right kidney, and then, bending more or less sharply to the left, crosses the second part of the duodenum. The hepatic flexure is thus in contact behind with the right kidney, anteriorly and above with the liver, and in close relation with the gall-bladder, duodenum, and pylorus. Although there may be some normal variation in the position of the flexure, it may be indicated by stating that, being about one inch above the lower border of the right kidney, which in the adult is within two inches of the iliac crest, it is about three inches above the iliac crest. Kuprijanoff found, when examining 101 subjects (8 embryos, 3 new-born infants, and 90 adults), that in 44·6 per cent the flexure was at the level of the 10th rib, in 23·4 per cent at the 11th, in 14·8 per cent at the 8th, in 6·8 per cent at the 9th, in 4·2 per cent at the 12th, and in 2·1 per cent at the 7th. These observations were, however, made on the cadaver and in the supine position with the abdomen open.

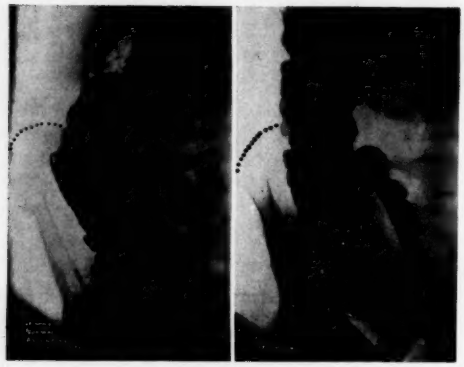


FIG. 388.—Showing the variation in position of the normal ascending colon and cæcum, in the erect and supine positions, 6 hours p.c.

McConnell and Hardman,[36] after radioscopic examination of a large number of middle-aged individuals who had never had any abdominal symptoms, came to the following conclusions: normally the diameter of the cæcum is only slightly greater than that of the ascending colon; the ascending colon has no lateral mobility; the hepatic flexure is almost a right angle, and is two and four inches above the iliac crest in the erect and supine positions respectively.

The hepatic flexure has, I think, little freedom of movement normally. The apparent alteration in the erect and supine positions during an opaque meal examination is largely due to the change in position of the proximal part of the transverse colon. Its fixation is due to its attachment to the posterior abdominal wall and to portions of the right kidney and duodenum consequent on the disappearance of its primitive mesentery. The 'right phrenocolic ligament' is merely the peritoneal reflection from the flexure to the posterior abdominal wall.

3. ABNORMAL MOBILITY AND PROLAPSE OF THE ASCENDING COLON AND CÆCUM.

VARIETIES AND DEGREES. (*See Figs.* 389-394.)

As in the evolution of the colon in man there is a '*gradual* fixation, embryologically and phylogenetically' (Tyrrell Gray), by fusion and obliteration of the mesentery, it is not surprising to find many variations depending on irregularities in the disappearance of the mesentery. Almost every variety and degree of abnormal mobility may be found. The mesentery may persist in its entirety. The mesentery

of the descending colon may disappear, that part of the bowel becoming fixed, while the remainder persists and rotation of the colon does not take place. But even if normal rotation has occurred, there may be irregularities in the disappearance of the mesentery of the ascending colon.

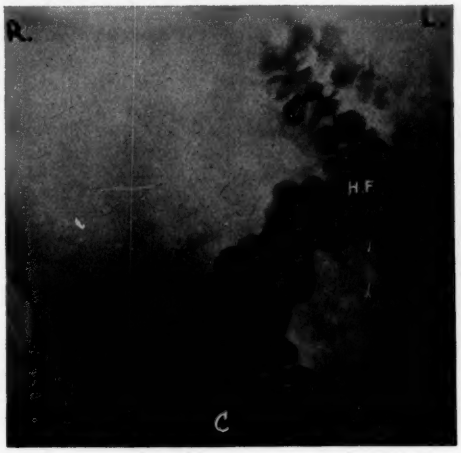


Fig. 389.—Showing non-rotation of the colon, in the erect position.

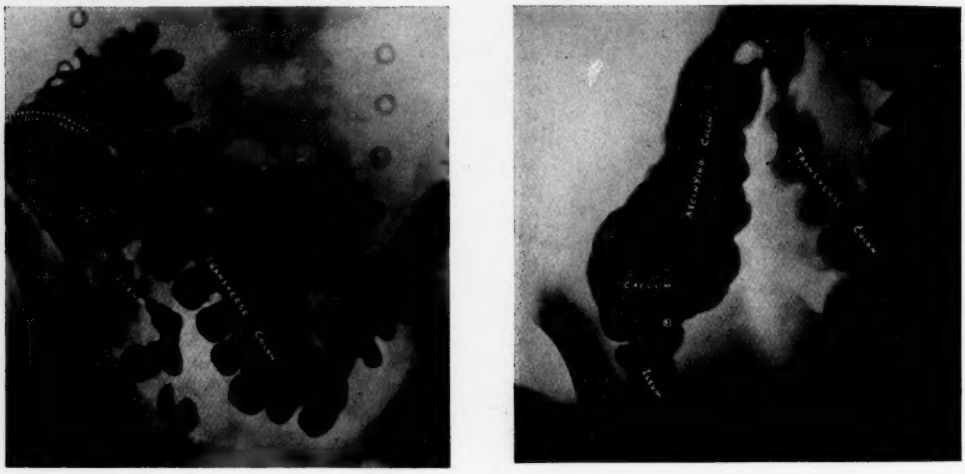
The mesentery of the whole length of the ascending colon may persist, and this part of the bowel be abnormally mobile and consequently prolapsed. This condition, which was present in 124 of the 242 cases under review, can be well demonstrated by the opaque meal. In the erect position the hepatic flexure may lie well below

Fig. 390.—Showing prolapse of the whole of the ascending colon and cæcum, in the erect position, 24 hours p.c. Note the wide-mouthed appendix.

Fig. 391.—Showing prolapse of the upper part of the ascending colon, in the erect position, 24 hours p.c.

the iliac crest, and the cæcum in the true pelvis. When the patient lies down, while the cæcum may remain in the pelvis the hepatic flexure can be easily moved up to its normal position under the liver. In addition to this vertical excursion, which is often as much as six inches, marked lateral mobility can also be easily demonstrated

On the other hand, absorption of part of the mesentery may have taken place, the remaining part of the ascending colon being abnormally mobile. Thus fixation was confined in 39 cases to the upper segment, in 20 cases to the middle segment, and in 14 cases the upper segment alone was mobile and had fallen down over the



FIGS. 392, 393.—Showing prolapse of the upper part of the ascending colon with partial non-descent of the cæcum, in the erect position in *Fig.* 392, in the supine position in *Fig.* 393.

lower. If the descent of the cæcum has been arrested, the mobile hepatic flexure may, in the erect position, be actually lower than the cæcum. In 11 cases the hepatic flexure alone was fixed in its normal position, the remainder of the ascending colon being mobile. In these cases the flexure was held in position not merely by the

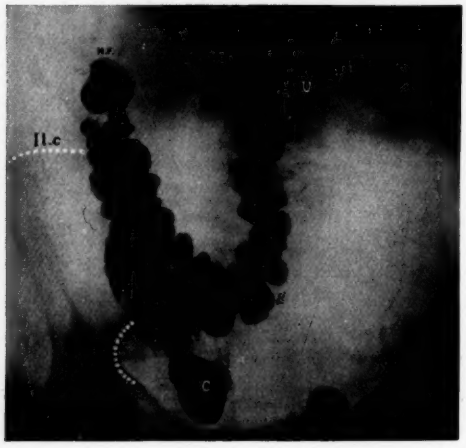


FIG. 394.—Showing prolapse of the cæcum alone, in the erect position.

peritoneal reflection but also by developmental membranes connecting it with the duodenum, gall-bladder, or liver.

In the majority of the remaining 34 cases, although the condition of the whole of the ascending colon could not be satisfactorily investigated through the gridiron incision employed, the abnormal mobility seemed to be confined to the cæcum alone.

This condition of 'cæcum mobile' represents the persistence of the infantile elongated type of cæcum.

In 25 cases the prolapse of an abnormally mobile cæcum was restricted by the presence of a 'controlling appendix', a complicating factor already referred to.

ANATOMICAL AND PATHOLOGICAL RESULTS.

The abnormally mobile ascending colon being attached to the posterior abdominal wall by a mesentery whose extent corresponds to that of the bowel which is mobile, "the full weight of the gut with its load is distributed along the narrow line of the posterior attachment of its mesentery, instead of being dispersed over a broad area sideways, as when no mesentery is present" (Waugh). This may result in the dragging out, in varying degrees, from the fascia of the posterior and lateral abdominal wall of a 'pseudomesentery' which is readily distinguishable from the true mesentery by its "opaque white surface devoid of blood-vessels", and by the definite 'white line' which marks its junction with the edge of the true mesentery. This false mesentery may be entirely absent, even when the whole of the ascending colon is abnormally mobile, but when present its extent usually corresponds to the extent of the bowel which is mobile. In this series it was found in slightly more than 50 per cent of the cases. It is without doubt an acquired condition. Waugh reports that "it is hardly ever found in children under twelve", although persistence of part or all of the true mesentery of the ascending colon was noted in 20 per cent of several hundred post-mortem examinations. While in 3 of the 5 of my cases under twelve years of age it was present, it was not nearly so extensive or so well-developed as in the older cases.

If, however, the mesentery of the upper and lower parts of the ascending colon has persisted while absorption has taken place in the middle part, this segment may be apparently bound down by an irregular opaque non-vascular membrane or band stretching from the outer side of the bowel to the posterior and lateral abdominal wall. This is the 'parietocolic membrane' described by Lane in 1903. McConnell and Hardman report having seen this frequently, in varying degrees, in new-born children and infants under two years of age; and it may represent one of the variations in the process of fixation of the bowel, being the result of irregular excessive fusion, thus resembling the mesosigmoid membranes. While it is most commonly seen about the middle of the ascending colon, it may be found opposite any part less mobile than elsewhere, and in my opinion is, in its fully developed state, an acquired condition, being a partial false mesentery drawn out at the point of maximum traction. Tyrrell Gray refers to a Jackson's membrane sometimes developing 'additional constricting bands', and H. M. W. Gray states that "quite substantial bands may be present which may be so short as to interfere with the movements of the colon". Both writers are evidently referring to this fascial structure, which differs both in appearance and origin from Jackson's membrane. I have in several cases seen the opaque, non-vascular fascial bands covered by a typical Jackson's membrane easily separable from them.

In cases where marked mobility and prolapse of the cæcum alone is present there may be recurrent limited volvulus of that part of the bowel.

The effect of the load in the abnormally mobile and prolapsed ascending colon and cæcum may extend to other parts of the bowel, and to organs with which it is directly or indirectly connected. Thus, if a Lane's membrane is present, an abnormally mobile cæcum may cause angulation of the ileum at the site of attachment of the membrane; but in remarkably few cases does this result in any degree of obstruction. Again, the drag of a loaded mobile cæcum on a 'controlling appendix' may result in kinking of that organ and thus predispose to attacks of inflammation. (The frequent association of the wide-mouthed infantile type of appendix with abnormal mobility of the ascending colon and cæcum has already been referred to, and the importance of this as a factor in the causation of certain forms of appendicitis will be discussed later.)

If a well-developed right intercolic membrane is present, there may be increased angulation at the hepatic flexure with aggravation of stasis in the ascending colon.

In cases of highly-placed splenic flexure, if the hepatic flexure, either alone or with the whole of the ascending colon, is much prolapsed, the transverse colon may be almost vertical in direction, and increased angulation at the splenic flexure with aggravation of stasis in the transverse colon result.

The pathological results of stasis on the wall of the colon do not require description, but will be referred to later when discussing the various forms of treatment.

The strain of the loaded ascending colon and cæcum is *transmitted to the other viscera* through the normal anatomical connecting links—for example, the mesentery of the small intestine, the transverse colon, the lesser omentum, the peritoneal reflections—but the transmission may also take place through the developmental membranes already described involving the duodenum, pylorus, gall-bladder, liver, and right kidney. In this series of cases I have seen definite evidence of transmission of the strain from the hepatic flexure or proximal part of the transverse colon directly to the liver in 13, to the gall-bladder in 13, to the duodenum in 6, and to the pylorus in 4; from the duodenum directly to the liver in 7, and to the gall-bladder in 20; from the hepatic flexure to the right kidney in 1. A ventral membrane involving the gall-bladder alone was seen in 26. The effect produced by these strains varies greatly, depending not only on the length and attachments of the membranes, but also on the relative position of the organs to which they are attached.

Thus, if the *gall-bladder* ventral membrane is neither too short nor dragged on by extensions to a mobile pylorus, duodenum, transverse colon, or hepatic flexure, there may be no interference with the function of the gall-bladder; but if any of these conditions is present there may be marked kinking at the junction of the gall-bladder and cystic duct. The importance of the resulting stagnation of the contents as a factor in the causation of gall-stones will be discussed later. As a secondary result, due partly to the interference with the evacuation of its contents, inflammatory adhesions may form in the vicinity of the gall-bladder, and it may be difficult to differentiate between developmental membranes and inflammatory adhesions which have arisen independently or in consequence of them.

The effect of the strain on the *pylorus* and *duodenum* also depends to a large extent on whether they prolapse or remain fixed in their normal position. In the latter case damage may be done at the point of attachment of the dragging membrane, and the importance of this as a factor in the causation of ulceration will be discussed later. The *stomach* and pylorus may, however, prolapse while the duodenum remains suspended by hepatoduodenal or cysticoduodenal membranes. The ptosed stomach becomes dilated and loses its functional activity; the pylorus takes up a vertical direction. The duodenum may also prolapse, and, although it may remain normal in size, there is usually some degree of dilatation. (It must, however, be remembered that abnormal mobility of the duodenum may be due to incomplete fusion of its mesentery with the posterior parietal peritoneum.) Marked dilatation of the first and second segments due to kinking at the junction of the second and third segments by the drag of membranes attached to that point was seen in 5 cases, but chronic duodenal dilatation is much more commonly due to the strain of the prolapsed ascending colon and cæcum being transmitted to the superior mesenteric vessels.

Chronic Duodenal Ileus.—The condition formerly known as 'acute dilatation of the stomach', and later as 'acute gastromesenteric ileus', has been usually attributed to an obstruction at the duodenojejunal junction. Relief from this acute condition may be obtained by elevation of the pelvis, or by placing the patient in the prone position, accompanied by gastric lavage. Condon[37] reports eight cases in which he obtained a cure by the injection of large quantities of normal saline into the peritoneal cavity, the patients being placed in Fowler's position, and the intestines being thus floated up. The condition seems to be due to a definite mechanical obstruction of the duodenum, not at the duodenojejunal junction, but at the point where the third part is crossed, and compressed, by the root of the mesentery.

The closely related condition of chronic duodenal ileus, although studied by many observers in France and America, had received little attention in this country

before the publication of Wilkie's first paper.[38] The normal duodenum is slightly compressed as it passes across the vertebral column under the root of the mesentery, and if the angle between the superior mesenteric vessels, which cross the third part at this point, and the vertebral column is lessened, then a degree of duodenal obstruction may result. This may be congenital. Wilkie refers to a patient operated on at the age of 7 whose symptoms began soon after birth, and in whom he found obstruction of the third part of the duodenum by the superior mesenteric artery due to malrotation of the gut. Dubose[39] and Downes[40] also have described cases of enormous dilatation in children evidently due to a congenital lessening of this angle. In the adult, however, the condition is apparently caused by more or less intermittent compression of the third part due to traction on the superior mesenteric vessels.

The most common cause of this is prolapse of the ascending colon and cæcum with traction through the ileocolic or right colic vessels. In this series of 242 cases moderate dilatation in 53 and marked dilatation in 26 was noted to be due to this cause. Of the latter group, in 23 the drag was through the ileocolic vessels, in 2 through the ileocolic and right colic vessels, and in 1, in which the upper part of the ascending colon alone was prolapsed, through the right colic vessels.

Another cause of traction may be the binding down of the terminal ileum by an extensive but short Lane's membrane. This condition was present in one case in this series in which the upper part of the ascending colon alone was prolapsed, and in two other cases in which there was no prolapse of the ascending colon or cæcum. I have also seen traction caused by masses of tuberculous glands in the mesentery of the small intestine, either by their weight or by torsion or distortion of the root of the mesentery. Wilkie refers to four such cases. In several other cases I have seen apparent shortening of the mesentery due to healed tuberculous lymphangitis along the ileocolic vessels. Wilkie also refers to the possibility of the drag of the small intestines if they are not supported on the pelvic floor, as in cases of tuberculous peritonitis.

Of the 26 cases in this series in which there was marked duodenal dilatation apparently due to compression by the superior mesenteric vessels, in 16 the condition was equally well marked in the first, second, and third segments; in 6 it was chiefly in the third segment; in 3 in the second and third segments; and in 1 in the first and third segments. This is not the experience of Wilkie, who states that although the dilatation may involve the first three segments it is usually most marked in the first. In this connection he suggests that the occurrence of an ulcer in the first segment may mask or modify the dilatation there. In only 2 of these 26 cases was there an ulcer of the duodenum, one inch beyond the pylorus, but in both the marked dilatation involved the first three segments equally. Wilkie has stated that the pylorus in these cases is 'usually dilated': this was seen in 10 of my cases, the dilatation being extreme in 4.

Now, if the duodenal dilatation were due to mechanical obstruction, one would expect it to be preceded by hypertrophy. Wilkie states that hypertrophy of the wall occurs, "the degree varying in different cases". I have, however, been impressed by the absence of any hypertrophy in the cases of moderate dilatation, and the thickening of the wall occasionally found in cases of marked dilatation may be accounted for by inflammatory changes resulting from the stasis. The purely mechanical explanation may thus be questioned. An alternative theory, with which Walton agrees, that the dilatation is due to inhibition of the duodenal wall, the result of sympathetic stimulation caused by increased mesenteric tension, will will be discussed later.

The loaded mobile ascending colon may also exert a drag on the *right kidney*. Southam,[41] writing on the fixation of the kidney, points out that the perirenal fascial sheath, which encloses the kidney, suprarenal, and perirenal fat, and is attached to the diaphragm, vertebræ, and transversalis fascia, is the most important factor in maintaining the kidney in position. He thinks that the amount of perirenal fat is of little importance, being practically absent in children, and that it serves chiefly as a protection from injury. Another factor of importance undoubtedly is the

intra-abdominal pressure, the loss of which is most seen in the maternal type of faulty body formation. In the female, again, the renal fossa is cylindrical in shape, being wider below than above, whereas in the male it is pear-shaped and narrower below. The position of the right kidney, however, is also influenced by other factors. Reference has already been made to the 'duodenorenal' and 'colicorenal ligaments', developmental membranes from the duodenum and hepatic flexure to the anterior surface of the right kidney, through which the strain of an abnormally mobile and prolapsed duodenum and ascending colon may be transmitted to the right kidney. In one of my cases, an unmarried female with a strong abdominal wall, the hepatic flexure, being attached by a well-developed membrane to the perirenal fascia, had in its descent dragged the kidney down almost to the iliac crest. Again, ptosis of the liver may have a direct action on the right kidney by means of the 'hepatorenal ligaments', developmental membranes connecting the under surface of the liver and the kidney. The prolapse of the kidney may be so marked that partial obstruction of the right renal vein occurs, giving rise to passive congestion with hæmaturia.

Waugh has reported that "even before operation it has been found that the right kidney was mobile in 79 per cent of all the cases". Of the 147 cases in this series in which the actual position of the right kidney was noted during the operation, it was normal in 32 per cent (18 males and 29 females, of whom only 3 had been pregnant) and prolapsed in 68 per cent (10 males and 90 females, of whom 36 had been pregnant). In 27 of the 63 cases of general visceroptosis the *left kidney* also was prolapsed.

The loaded cæcum, lying as it so often does in these cases in the pelvis, may cause or aggravate a displacement of the *uterus*. This will be referred to later when describing the symptoms and giving the clinical results.

I have thus described in detail the anatomical and pathological effects of the drag of the loaded ascending colon, either directly on the posterior abdominal wall or more or less remotely on the neighbouring organs, either through normal connecting links or through developmental membranes. It would, however, be a profound mistake to treat the problem of stasis, either in the ascending colon or elsewhere in the alimentary tract, as purely a 'problem in mechanics' (Waugh).

4. MECHANICAL AND INHIBITORY THEORIES OF STASIS.

Waugh, in describing the mobile ascending colon as "mechanically inefficient for carrying out its normal function", attributes the failure of peristalsis to the abnormal mobility of the bowel. "The wave of peristalsis starting in its lowest segment will expend its energy partially on the mobile segment of the gut immediately above it, and partially upon its contents, instead of entirely upon its contents as it would if the gut were fixed . . . the tendency to progressive overloading is established through the wasteful expenditure of the wave of peristalsis in moving the gut as well as the load". He states further that constipation is the final refusal of the bowel to perform "its natural function under mechanical conditions badly suited to it". This explanation of peristaltic failure is not convincing. For example, it would not explain why peristalsis improves when the "broad plateau of the gravid uterus supports the prolapsed colon", or when the supine position is assumed. In both these circumstances, although the mesenteric drag has been relieved, the ascending colon remains "a mobile and not a fixed viscus". The method by which food is passed along the intestinal canal is much more complex than he infers.

Normal intestinal movements are of two varieties: peristaltic waves of contraction and dilatation travelling along the intestine and driving the contents on; and rhythmic or segmentation movements which, by forming a number of alternately contracted and dilated areas, each of which is divided into two by the next movement, mix the contents but do not move them on.

While there is no doubt that the peristaltic movements are affected by extrinsic nerves, the parasympathetic increasing, the sympathetic inhibiting them, Tyrrell Gray is of the opinion that "the contraction elicited by the mechanical stimulus of intestinal contents is itself responsible for exciting mechanically the intramural

inhibitory sympathetic fibres to the segment immediately in front". He holds also that the rhythmic movements "serve to maintain constant contractions on the intestinal contents, and, when these are accumulated in sufficient bulk, to elicit that mechanical pressure stimulus which initiates peristalsis".

Now, rhythmic waves, which are normally continuous, become accentuated by stimulation of the parasympathetic. Thus vagus stimulation resulting from the entry of food into the stomach results in increased rhythmic contractions in the ascending colon and elicits peristaltic waves with mass movement (the 'gastrocolic reflex' of Hurst). Mass movement is also initiated by the act of defæcation by a reflex stimulation of the parasympathetic through the lumbar centre. Should the peristaltic contraction be too feeble to cause a mass movement, there may be a movement slightly forward followed by a slipping back, the so-called 'antiperistalsis'. The accentuation of the mechanical stimulus by the arrival of additional contents, or the increase in rhythmic contractions by the renewal of the parasympathetic stimulation, may result in successful peristalsis.

In studying derangement of normal intestinal movements, Tyrrell Gray has stated that the cessation of movement when the abdomen is opened under general anæsthesia does not occur under spinal anæsthesia, the inhibition in the former case being due to a reflex sympathetic stimulation. He has also demonstrated that while manipulation of the wall of the bowel inhibited the tone of the part involved, traction on the mesentery inhibited the whole area supplied by the mesenteric nerves so stimulated. Inhibition from inflammatory lesions of the peritoneum is well recognized. Inhibition from the mechanical stimulation induced by the drag on the mesentery must also be accepted. Tyrrell Gray answers the possible criticism that such stimulation should affect equally the parasympathetic nerves, by referring to the difficulty in experimentally stimulating the vagus in its efferent course, and to the increasing predominance of the sympathetic, and the ease with which it can be stimulated, in the lower portions of the intestinal tract. This efferent stimulation, induced by a drag on the mesentery, resulting in local inhibition of the bowel supplied, is accompanied by afferent stimulation with general partial intestinal inhibition and spasm of Keith's 'sphincters' which divide the alimentary tract into a number of neuromuscular segments.

Tyrrell Gray agrees with Waugh that the ascending colon is normally sessile, and being supported by its attachment to the posterior abdominal wall, does not exert any traction on its mesocolon, in which lie the vessels and nerves which control its nutrition and movements. If, however, owing to abnormal mobility of the bowel, the strain is transferred to the mesentery, there is a mechanical stimulation of the sympathetic nerves, with inhibition and derangement of the normal intestinal movements. Accompanying this there is also spasm of the neighbouring Keith's 'sphincters'. The consequent overloading in the ascending colon and cæcum increases the mechanical stimulus, accentuates the traction on the mesentery, and thus results in a vicious circle.

Acknowledging that abnormal mobility of the ascending colon may be symptomless, Tyrrell Gray suggests that the initiation of symptoms is due to a relative increase in mesenteric tension, the result of either a loss of support or an abnormal loading of the bowel.

In noting the possibility of angulation of the terminal ileum at the site of attachment of a Lane's membrane, with or without abnormal mobility of the cæcum, I remarked on the extreme rarity of any degree of actual obstruction. Tyrrell Gray, discussing the possible association of ileal stasis with the presence of a Lane's membrane, does not accept the theory of mechanical obstruction at this point. He refers to the frequency with which acute kinking or distortion of the small intestine is seen, for example, in cases of healed tuberculous peritonitis, without any degree of obstruction, and points out how even partial mechanical obstruction is invariably accompanied by hypertrophy of the proximal intestine—a condition he has never seen in connection with a Lane's membrane. He is of the opinion, however, that the membrane may, by causing increased tension in the mesentery of the terminal ileum, especially in the erect position, result in stimulation of the sympathetic nerves

distributed to the ileocæcal region, with inhibition of the normal rhythmic and peristaltic contractions, and at the same time spasm of the ileocæcal sphincter, and ileal stasis.

The association of cæcal stasis with a Lane's membrane may be similarly explained, as the area inhibited may include the cæcum as well as the terminal ileum. The 'controlling appendix' and the mesosigmoid membranes may also be capable of inducing inhibitory stimulation of the sympathetic nerves of the terminal ileum and pelvic colon by causing traction on their mesenteries.

It has already been noted that chronic duodenal dilatation is apparently due to compression of the third part of the duodenum by traction on the root of the mesentery from various causes, but that hypertrophy of the duodenal wall is rarely if ever present. It seems possible therefore that the dilatation is not the result of partial mechanical obstruction, but is due to sympathetic stimulation caused by increased mesenteric tension. In addition to inhibition of the duodenum there would be spasm of the pyloric and duodenojejunal sphincters, which may account for the fact that pyloric dilatation, usually accepted as retrograde, may not be present even when the duodenal dilatation is extreme.

I have already pointed out the possibility of the ventral membrane causing kinking at the neck of the gall-bladder with consequent stagnation of its contents. Tyrrell Gray remarks on the frequency with which the gall-bladder in cases of visceroptosis is found to be tense, or even distended, and difficult to empty with even firm pressure. In the absence of any obvious obstruction or kinking, he thinks that this may be accounted for by a reflex sympathetic stimulation resulting from abnormal mesenteric tension elsewhere.

Gastric stasis, so commonly seen in cases of right-sided ptosis, may also be due to prolonged pyloric closure with inhibition of the pyloric half of the stomach, the result of a similar reflex sympathetic stimulation.

Thus, although purely mechanical kinking or traction may be an important etiological factor in the production of stasis in the cæcum, ascending colon, gall-bladder, duodenum, and stomach, associated with abnormal mobility and prolapse of the ascending colon and cæcum, it seems more reasonable in many cases to attribute the condition of stasis to inhibition due to sympathetic stimulation resulting from abnormal mesenteric tension, either local or remote.

II. CLINICAL SYMPTOMS AND SIGNS.

As visceroptosis may be unaccompanied by any symptoms, and is indeed often found in those in perfect health, great care must be exercised in deciding that the clinical condition in any particular case is due to visceroptosis. It is not sufficient to be satisfied that the condition could be accounted for by visceroptosis; one must exclude the possibility of its being due to other pathological conditions. In emphasizing this, Conybeare[42] points out that "to the lay mind the fact that the viscera are dropped or misplaced offers a reasonable explanation to account for symptoms of any and every kind; moreover, it is an idea which it is much easier to implant than to eradicate".

X-ray investigation by the opaque meal or enema, which must be looked on as merely a single link in the diagnostic chain, is of great value in determining not only the shape, position, and degree of mobility of the viscera but also their functional activity. It is, however, a dangerous method of diagnosis unless wisely used, and is meaningless unless the findings are intelligently interpreted in conjunction with the clinical history. For example, there are many variations in the mode of preliminary preparation, in the type of meal, and in the restrictions imposed on the patient during the period of examination. The mental condition of the patient during the examination also is

of great importance. The investigation must be thoroughly and systematically carried out, the patient being throughout under the control of the observer. Screening is undoubtedly the most effective method, and films must be looked on merely as more or less disconnected illustrations of the story presented by the radiologist to the surgeon. Screening must be done at will in various positions, and visualized palpation should be employed if possible.

In spite of technical precision and careful observation, errors may occur. For example, in examining the duodenal bulb the radiologist may mistake deformity due to reflex spasm or periduodenal adhesions or membranes for an ulcer. Or, again, neglecting to fill the bulb completely or to view it from different angles, he may wrongly infer that there is no ulcer because he has failed to visualize any duodenal deformity. If the radiologist either confirms or refuses to confirm the surgeon's suspicion of the presence of an ulcer, and if, when the abdomen is opened, there is no external, peritoneal evidence of an ulcer, a difficulty arises. Is the surgeon justified in opening the duodenum to confirm or refute the opinion of either the radiologist or himself that an ulcer is present? If a thorough search is impossible, owing either to local conditions or to the general condition of the patient, should the surgeon, relying on the radiologist's opinion, presume the presence or absence of an ulcer and perform the appropriate operation? The value of the X-ray investigation is greatly increased if there are close co-operation and mutual confidence between a radiologist who follows his cases to the operating theatre and a surgeon who is capable of appreciating the radiological interpretation. In this way alone can the problem of how far the surgeon should rely on the radiologist's opinion be solved, and radiological investigation prove useful in determining the pathological cause for the clinical condition.

SYMPTOMS.

Those patients whose clinical condition is attributable to abnormal mobility and prolapse of the ascending colon and cæcum show, as a rule, in their early stage somewhat similar characteristic symptoms, which, however, may become obscured as the results of the prolapse of and consequent stagnation in the colon become more definite.

The *onset* may be indefinite, but usually occurs at some definite time when the potential disability—ptosis—becomes an actual disability—stasis—the balancing factors being disturbed either by a weakness developing or an extra call being made. Thus the onset of symptoms in the virginal type of faulty body formation usually occurs at puberty, and is more often found in girls than in boys. The apparently healthy girl or boy being sent to a public school may be improperly fed, and may pay more attention to strenuous games, for which she or he is not fit, than to the daily evacuation of the bowels. In the maternal type the onset usually occurs soon after a pregnancy when the patient returns to active life.

Waugh's explanation of the method of the onset of stasis cannot be accepted. He holds that the abnormally mobile ascending colon, 'mechanically inefficient' from the first, exhibits its maximal inefficiency at adolescence

when it has "achieved its largest size and contains its heaviest load". As "no proportionate strengthening of the primitive mesentery" has accompanied this increase in the load, stasis occurs. Tyrrell Gray, on the other hand, attributing the onset of stasis to any factor increasing the normal tension in the mesentery, is of the opinion that this usually results from loss of support from the abdominal wall, but may be due to overloading of the bowel resulting from various conditions—for example, appendicitis, which would be accompanied by inhibition of the ileocæcal area.

The *early symptoms* are usually described by the patient as 'stomach trouble', 'indigestion', or 'dyspepsia', as there is generally discomfort after food with more or less distressing flatulence. At the end of a variable time, if dietetic and medicinal treatment fail, there occurs the onset of pain. This pain is at first, and often throughout the whole course of the illness, characteristic. It is constant in type and location for each individual, but is very erratic as to its times of appearance and duration. Thus it may be present for one or more days, with free intervals of varying length; it may, on the other hand, last less than a day. Waugh, in emphasizing this 'constant inconstancy', suggests that it is because of this that many of these patients are, quite unjustly, considered to be neurotic.

Now, although what is commonly called 'dyspepsia' may occasionally be due to pathological changes in the gastric mucous membrane resulting from indiscretions in diet, pyorrhœa alveolaris, etc., in most cases the stomach itself is not at fault, it is 'more sinned against than sinning', and the cause for the so-called 'stomach symptoms' must be sought elsewhere.

Reflex or Acid Dyspepsia.—Tyrrell Gray, referring to the difficulty experienced by physiologists in exciting the normal secretory and motor function of the vagus by electrical stimulation, suggests that these are normally elicited reflexly by afferent impulses. He is of the opinion that the afferent vagus stimulus responsible for the sensation of hunger is not due, as has been generally believed, to contractions of the stomach but to engorgement of the gastric and pancreatic glands during the resting period, which results in an efferent response to gastric glands and muscle. Stimulation of the vagus results in secretory and motor activity in the cardiac half of the stomach. Hydrochloric acid is secreted by the oxyntic cells, and by the contraction of the circular and longitudinal muscle fibres the lumen is diminished, by the contraction of the oblique fibres the fundus is raised, and the contents are forced into the pyloric half of the stomach. This excites a mechanical pressure stimulus which results in peristalsis. Relaxation of the pylorus is due to the presence in the duodenum of alkaline pancreatic secretion which also is the result of vagus stimulation. Bolton and Salmond[43] have shown that, in addition to regulating the evacuation of the stomach contents, the pyloric sphincter in the early stages of digestion prevents regurgitation from the duodenum, but that as digestion proceeds it may by relaxing, independently of gastric peristalsis, permit of regurgitation by normal antiperistalsis in the duodenum.

If there is, for any reason, a reflex stimulation of the sympathetic, only the pyloric half of the stomach is affected, the cardiac half not being supplied with sympathetic inhibitory nerves. The result is inhibition of peristalsis with prolongation of pyloric closure. The latter is accentuated also by the

diminution of pancreatic and the increase of adrenal secretion. The glands at the cardiac end, not being inhibited, continue to secrete hydrochloric acid, the percentage of which is thus raised above the normal, partly by delayed evacuation of the gastric contents and partly by absence of duodenal regurgitation. While a simple hyperchlorhydria due to actual over-secretion associated with over-action of the vagus may occur, it is rare, hyperchlorhydria being usually due to reflex sympathetic inhibition.

To the condition of intermittent discomfort or even pain after food, not dependent on the kind of food and accompanied by flatulence and by hyperchlorhydria, may be given the name 'reflex' or 'acid dyspepsia'. The appendix and gall-bladder are the most commonly accepted foci of irritation responsible for the reflex stimulation. Moynihan,[44] describing the condition of pyloric spasm, gastric stasis, and hyperchlorhydria which is curable by the removal of an inflamed appendix, and which he has called 'appendicular dyspepsia', affirms that it is accompanied by acute congestion of the mucous membrane of the antrum pylori. Other observers, however, finding no changes in the gastric mucous membrane, regard the condition as purely reflex. Again, gall-stones which may remain in the gall-bladder for years without producing biliary colic are not so 'silent' as is generally supposed, being very often the cause of repeated attacks of reflex dyspepsia, the reflex stimulation being set up by an alteration in the position of the calculi, or by a slight attack of cholecystitis. The inhibitory stimulus may, however, be found not in any pathological focus of irritation but in increased mesenteric tension. The condition of abnormal mobility of the ascending colon and cæcum is undoubtedly a common cause of inhibitory stimulation, and that the condition of acid dyspepsia and gastric stasis may be attributable to this is proved by their disappearance after removal of the abnormal mobility by fixation of the colon.

Although spasm of the pylorus may in its inception be protective, it is undoubtedly an important factor in the production of *pain*. The relationship between the occurrence of the characteristic pain already referred to and the ingestion of food may be explained by the effect produced on the ascending colon by taking food. I have already noted that, by the 'gastrocolic reflex' of Hurst the entry of food into the stomach is followed by an attempt, successful or not, on the part of the cæcum and ascending colon to pass on their contents. If this peristaltic activity is unsuccessful, the load in the cæcum and ascending colon remains unaltered until it is increased by the arrival, usually about three hours later, of an additional load.

Waugh attributes the occurrence of pain soon after food in cases of abnormal mobility of the ascending colon to its unsuccessful peristaltic effort, with the resulting drag on its mesentery, and the transmission of the drag to the duodenum and pylorus should developmental membranes connecting these structures be present. Pain several hours after food is due to the effect of the addition to the load in the ascending colon and cæcum. On the other hand, if the peristaltic effort is successful, then ingestion of food, by indirectly helping to shift the load, may relieve the pain. The relief which may follow an evacuation of the lower bowel may be similarly explained, as the act of defæcation excites a reflex stimulation of the parasympathetic. The relief

obtained by lying down with the pelvis raised is due to the removal of the mesenteric drag and the resulting increased peristalsis. The 'erratic chronology' of the pain, its absence for days at a time, its unexpected recurrence, may be explained by the fact that the condition of the cæcum and ascending colon, and the strain exerted by their contents, vary very much in the same individual day by day. The pain is inconstant because of the varying strain produced by the varying load.

An additional factor in the association between the ingestion of food and the onset of pain is to be found in the duodenal dilatation so frequently present in these cases. There can be no doubt that the entry of food into the dilated duodenum, with its more or less prolonged residence there, may be an important factor in the production of pain in a similar way to that described by Waugh.

I have already indicated that the characteristic pain after food associated with abnormal mobility of the ascending colon is in some cases relieved by taking more food, and is also amenable to change of position from the vertical to the horizontal. That this so-called 'hunger pain', described by Moynihan in 1905 as occurring in the majority of chronic duodenal ulcers, is not necessarily indicative of duodenal ulceration cannot, I think, be doubted. Waugh states that, although 97 of the 518 patients on whom he performed a right colopexy had 'hunger pain', none of them had a duodenal ulcer. I cannot, however, understand his refusal to admit that similar relief is often experienced in cases of duodenal ulceration. He asserts that " a duodenal ulcer, in being soothed by active movement and arrival of foreign material in the duodenum, is made to exhibit characteristics which ulcers elsewhere do not display ". The explanation that this relief was due to closure of the pylorus following the ingestion of food, the ulcer being in this way relieved from the irritation of the stomach contents, is not now accepted in the light of opaque meal examination. This has proved beyond all doubt that in cases of duodenal ulceration the entry of food into the stomach is followed by an abnormally rapid evacuation, the 'duodenal rush', and that the gastric contents are actually passing over the ulcerated area at the time the relief is experienced. Before evacuation is complete, however, the pylorus is again closed and the pain returns. Thus 'hunger pain' can only be explained by spasm of the pylorus, and the relief obtained is due to the relaxation of the pylorus which in this case follows soon after the entry of food into the stomach.

Waugh is so confident in his ability to discriminate between the characteristic pain associated with abnormal mobility of the ascending colon and that due to gastric or duodenal ulceration that he does not examine the stools for occult blood, nor does he make use of opaque meal examination. He states that he has not been penalized for this omission by the unexpected discovery of an ulcer at the operation. I cannot help thinking that he is taking uncalled-for risks, as a thorough examination of the stomach and duodenum at the operation may be unwise, if not actually impossible. An ulcer may be very insidious in onset, and an opaque meal examination might demonstrate its presence even in its earliest stage. In excluding the possible presence of ulceration, either gastric or duodenal, one must depend chiefly on the character of the pain. If there is an ulcer, the patient is usually never

absolutely free from pain, there are no free intervals, the pain comes on after every meal, on successive days, and benefit is almost invariably obtained by restrictions of diet, which are usually self-imposed. Contrast this with the inconstant erratic pain which is not dependent on the kind of food, which is associated with abnormal mobility of the ascending colon.

Recurrent attacks of pain in the right iliac fossa, usually quite independent of food, occur in many cases, and seem to be due to recurrent over-distention or actual volvulus of the cæcum. A dragging pain in the right lumbar region, associated with prolapse of the right kidney, is also frequently present. Should the patient suffering from these symptoms become pregnant, the symptoms often disappear, only to reappear soon after the termination of the pregnancy. The temporary relief is due to the removal of the drag on the mesentery by the lifting up of the ascending colon by the pregnant uterus as it rises from the pelvis.

In the *later stages* the frequent occurrence of 'bilious attacks' is due to stasis and consequent pathological changes in the dilated duodenum. In the later stages also there is loss of appetite and increasing flatulence, often of distressing severity. In the early stages there may have been constipation, but in the later stages this is almost invariably present, and is often extreme and obstinate. It may be accompanied by attacks of diarrhœa with mucus in the stools. Waugh considers that the constipation is the result of the final refusal of the ascending colon to " perform its natural function under mechanical conditions badly suited to it " ; Tyrrell Gray explains it as a " derangement of the nervous impulses responsible for the normal intestinal contractions " due to abnormal mesenteric tension.

Menstrual disturbances are common, and seem to be due to the presence of the loaded cæcum in the pelvis.

Finally, after a varying period, toxic phenomena occur, such as progressive wasting, pigmentation of the skin, chronic mastitis, and loss of physical and mental vigour. Various secondary or end-results which may reasonably be attributed to the persistence of abnormal mobility and prolapse will be described after referring to the clinical types into which it is convenient to divide these cases.

The Mental Factor in Visceroptosis.—The difficulty of appreciating the relationship between visceroptosis and neurasthenia, of deciding whether in neurotic subjects who have gastro-intestinal disorders the neurosis is primary or secondary to prolonged intestinal derangement, cannot well be exaggerated. I cannot understand the dogmatic opinion of Stoddart[45] that " the most frequent cause of visceroptosis is dilatation of the stomach : without this the colon cannot fall : by far the commonest cause is anxiety, normal or morbid : and this is the etiological factor we should try to combat ". Gastroptosis, with or without dilatation, is not the cause of visceroptosis : it may be part of that condition, but is more frequently secondary to right-sided ptosis. Thus, although in my series of 242 cases there was right-sided ptosis in all, in 110 the position of the stomach was normal.

Langdon Brown[46] supports Craven Moore's opinion that in cases of reflex dyspepsia there must be, in addition to a focus of irritation, an

increased susceptibility of the reflex nervous mechanism, and that the nearer the focus is to the pylorus the less unstable need the nervous system be. Remote foci—for example, ileocæcal lesions—may remain latent until the susceptibility becomes greatly exaggerated, and the more unstable the nervous system the less the irritative stimulus required. Against this it may be argued that the nervous system of many patients with visceroptosis, who suffer from the effects of abnormal mesenteric tension already described, is absolutely normal.

It cannot, however, be questioned that prolonged mental strain, anxiety, or grief may, by causing excessive stimulation of the sympathetic system with general inhibition of the gastro-intestinal tract, diminution of salivary, gastric, and pancreatic secretion, and increased adrenal output, result in symptoms similar to those under discussion. Should visceroptosis also be present in these cases, one must resist the temptation to attribute the symptoms to this instead of to the psychic cause. Should secondary changes associated with visceroptosis supervene, the fact that they are really psychical in origin must not be forgotten. Reference has already been made to Netschajew's suggestion that ptosis may be due to inhibition of the sympathetic nerve-supply of unstriped muscle fibres in developmental membranes, resulting in their relaxation.

Tyrrell Gray endeavours to differentiate between sympathetic inhibition due to psychical causes and that due to abnormal mesenteric tension, by suggesting that in the former there is in addition vagus inhibition. Deficiency in the afferent vagus stimulus, with absence of the sense of hunger and loss of appetite, would result in diminution of motor and secretory activity in the cardiac half of the stomach, with deficiency of hydrochloric acid and more marked stasis and dilatation. This argument is, however, not convincing, as the sympathetic inhibition caused by abnormal mesenteric tension may also result in increased adrenal and diminished pancreatic secretion with deficiency in the afferent vagus stimulus.

The problem can be solved only by a careful investigation of the clinical history in each case. Thus, if the abnormal mental condition of depression, introspection, emotion, exaggeration of symptoms, 'nervousness', etc., precede the development of any definite physical abnormality, the etiological factor is evidently psychical. In some of these cases a very definite psychical disturbance can be found—for example, domestic or financial anxiety, artificial menopause following pelvic operation, etc.

On the other hand, if a patient had suffered from acid dyspepsia, flatulence, recurrent attacks of right-sided abdominal pain, constipation, mucous colitis, menstrual disturbances, etc., before any mental abnormality appeared, one would be justified in considering the mental condition as secondary to the primary physical disability.

That toxic conditions due to visceroptosis may profoundly affect the nervous system cannot be doubted. (That many subjects with visceroptosis do not exhibit any mental abnormality is no argument against this, as visceroptosis may be unaccompanied by any symptoms.) This secondary mental condition in my opinion differs in some respects from that seen in cases of primary psychical disturbance. The patient, instead of being apathetic and depressed,

is more often irritable, 'disgruntled', disappointed, or actually annoyed at the loss of mental control and vigour. But even though one may feel sure that the mental condition is secondary, it is an exceedingly difficult problem to judge how profoundly the nervous system may have been affected and yet be amenable to improvement by the correction of the primary condition. If the patient is exceedingly anxious to regain a normal mental status, the outlook is good. But if there is a feeling of hopelessness, especially if secondary results such as mucous colitis have supervened, operative treatment may result in little mental improvement.

In cases of psychical origin in which secondary physical changes have supervened one may be greatly tempted to operate, hoping not only to relieve the physical but at the same time to improve the mental condition. While the former is usually successful, the latter is almost invariably unsuccessful.

SIGNS.

The signs vary greatly, the variation depending on the extent and degree of the mobility and prolapse of the ascending colon and cæcum, on the extent and distribution of the developmental membranes present, and on whether there is ptosis of other viscera in addition.

The difficulty of determining what constitutes an abnormally low

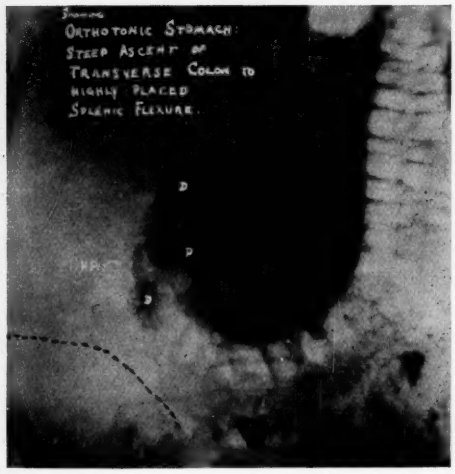


Fig. 395.—Showing a normal, orthotonic stomach, in the erect position.

Fig. 396.—Showing an abnormally low, orthotonic stomach, in the erect position.

position of the viscera has already been referred to. Moody, Van Nuys, and Chamberlain[47] report that when examining 600 healthy adults the stomach as shown by the opaque meal in the erect position reached below the interiliac line in 80 per cent. While there is considerable normal variation in its position, one can say that any stomach whose greater curvature as shown by the opaque meal in the erect position is more than one inch below the interiliac line is abnormally low. Any prolapse short of this may be considered normal. (*See Figs.* 395–398.) In all the cases in this series in which

the prolapse was limited to the ascending colon and cæcum the position of the stomach was normal. In the 69 cases in which the stomach and right kidney were also prolapsed the former reached from one to three inches below the line in 54, and to more than three inches in 15. In the 63 cases in which there was general visceroptosis it reached from one to three inches below the line in 26, and to more than three inches in 37. In 10 the greater curvature was actually more than six inches below the line. The stomach is thus very frequently abnormally low, and may lie almost completely in the pelvis. Even when low it may remain orthotonic, but there is usually some degree of dilatation and some delay in emptying. Splashing can thus readily be detected, and one is apt to come to the erroneous conclusion that there is pyloric stenosis.

The ascending colon is usually much dilated, and may be palpated in the right iliac fossa. There is usually diffuse tenderness over the right side of

FIG. 397.—Showing an abnormally low, hypotonic stomach, in the erect position.

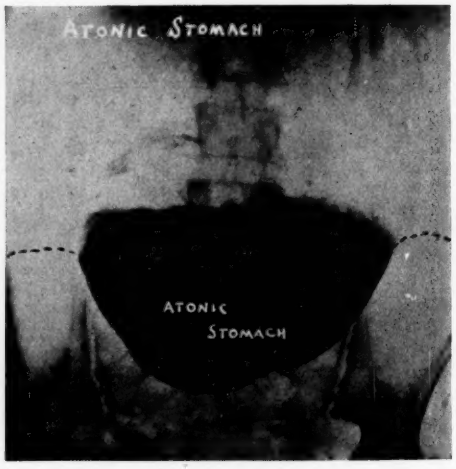


FIG. 398.—Showing an abnormally low, atonic stomach, in the erect position.

the abdomen, chiefly in the iliac region. On dragging on the prolapsed bowel a very definite 'drag pain' may be induced which is referred to the right hypochondriac or epigastric region, or to the left hypochondriac region if there is a long vertical transverse colon and a highly-placed splenic flexure.

The right kidney can usually be easily palpated, often in its full extent, and it may be so low as to be liable to crushing between the ribs and the iliac crest in stooping. If there is general visceroptosis, the left kidney may also be palpable.

The retroflexion and enlargement of the uterus so often present can be easily noted.

If the liver is ptosed, there is usually some rotation, and the right lobe projects below the costal margin, the projecting portion being sometimes separated from the remainder by a sulcus.

III. CLINICAL TYPES AND SECONDARY RESULTS.

CLINICAL TYPES.

It is convenient to classify the cases under review into various types depending chiefly on the type and location of the pain.

1. **The Gastric Type** (42 cases).—These patients have generally been previously treated for 'dyspepsia', 'chronic gastritis', 'gastric dilatation', or 'gastric ulcer'. The pain is usually located in the epigastrium, but if, owing to a marked prolapse of the hepatic flexure, there is a long vertical ascent of the transverse colon to a highly-placed splenic flexure, it is felt in the left hypochondrium. It occurs soon after food, always within an hour, but not after every meal or every day. It does not depend in any way on the kind of food taken, and these patients have usually given up dieting, as they find that they are as likely to be well when eating anything and everything as when on a strict diet. There is rarely nausea, and almost never vomiting. This pain thus differs in several respects from that of a gastric ulcer, which does not show this erratic behaviour and can be lessened by dieting. Other possible foci of irritation—for example, gall-stones, an inflamed appendix—must be excluded before attributing it to abnormal mesenteric tension due to abnormal mobility of the ascending colon. In addition to the pain there is always very marked flatulence; this may be so extreme that the patient is unwilling to appear in public or even to visit friends. So-called 'bilious attacks' are neither so severe nor so frequent as in the next type.

2. **The Duodenal Type** (32 cases).—Many of this type have been previously treated as 'duodenal ulcer'. The pain is usually located in the right hypochondrium. It comes on from two to four hours after food, but not after every meal or every day. It does not depend on the kind of food taken. It is dull or boring in character, and often has the characteristics of the so-called 'hunger' or 'food pain' in that it may be relieved by taking more food. It may, however, be increased by taking more food, or again, be relieved by the bowels moving. An additional factor in the production of the pain is the loading of the dilated duodenum, and it is in this clinical type that duodenal dilatation is most commonly found. In only 4 of the 32 cases was the duodenum noted as normal; in 14 the dilatation was 'moderate' and in 14 'marked'. The frequency of the occurrence and the severity of the 'bilious attacks' in this type (they were a prominent feature in 18) are undoubtedly explained by the frequency with which duode al dilatation was present. In some the attacks could be aborted in the early stages by the patient assuming the prone position; but in the later stages, when the dilatation had become permanent, the adoption of this posture had no effect. In the severe cases of this type the condition approached that of 'chronic duodenal ileus'.

3. **Right Iliac Fossa Type** (109 cases).—In this type there is a characteristic pain in the right iliac region. This is usually merely a severe discomfort. It is very erratic in its times of appearance, and there may be fairly long intervals of freedom. It has no definite connection with meals, but almost invariably the patient notices that it is more apt to be present if the bowels are not moving regularly, and that it is often relieved by an evacuation. In a majority of these cases there is obstinate

constipation; in some cases there are frequent attacks of diarrhœa with mucus in the stools. I agree with Waugh that this pain is due to recurrent over-distention of the cæcum, which also, by causing increased mesenteric tension, accounts for the reflex dyspepsia which may be present. In all cases of this type the cæcum was found to be dilated; in many the dilatation was very marked. It was abnormally mobile and prolapsed in most, but in 25 was suspended by a 'controlling appendix', and in 1 was partially undescended. Recurrent attacks of severe pain in the right iliac region occasionally occur in this type of case. These are invariably associated with marked mobility and prolapse of the cæcum alone, the ascending colon being normally fixed, and they seem to be due to recurrent limited volvulus of the cæcum, a condition that has occasionally been actually noted at operation.

As many of the patients had been previously considered to be suffering from 'chronic appendicitis' (about which more will be said later), the condition of the appendix as found at operation is of interest. In none was there any evidence of active inflammation, either acute or subacute. In 21 it was absolutely normal; in 40 the only abnormalities were the open mouth and the fæcal contents; in 25, being fixed to the iliac fossa by developmental membranes, it was acting as a 'controlling appendix'; in 9 obliterative changes had taken place; in 4 true inflammatory adhesions were present, which were probably the result of distortion by previously existing developmental membranes; in 10 the appendix had already been removed.

Removal of a 'choked appendix' will do away with attacks of true appendicular colic. Removal of an appendix deformed by developmental or inflammatory membranes will obviate the danger of acute inflammation. Removal of a 'controlling appendix' may relieve local intestinal stasis and reflex dyspepsia by removing the inhibitory stimulus, but the improvement can be only temporary, as, by allowing the cæcum to prolapse, further stasis will follow. Thus in this type of case simple removal of the appendix is not sufficient. That in cases of recurrent attacks of pain in the right iliac region the appendix may be unjustly blamed and its removal followed by no permanent improvement is shown by the fact that the ten patients of this right iliac fossa type whose appendices had already been removed without amelioration were all completely cured after the performance of a colopexy, partial or complete.

4. **The Renal Type** (6 cases).—In these cases there were attacks of pain somewhat similar to Dietl's crises, but unaccompanied by any variation in the quantity of urine passed. In all, the right kidney was prolapsed, and in one a well-marked developmental membrane from the prolapsed hepatic flexure to the perirenal fascia indicated clearly how the prolapse of the kidney had been brought about. The attacks of pain seemed to be due to partial obstruction of the right renal vein, with passive congestion and, in three cases, hæmaturia.

Waugh classifies some of his cases under the heading '*biliary type*'. In these there have occurred one or more attacks similar to biliary colic, but in none of them were calculi found at operation. Such attacks have been noted in many of my cases of both the gastric and duodenal types, but as they were additional to the other symptoms I prefer not to differentiate them

into a separate category. Although they seemed to be due to kinking at the neck of the gall-bladder by the ventral membrane, with perhaps in addition stagnation and increased viscidity of the bile, the possibility of inhibitory stasis by reflex sympathetic stimulation must be borne in mind. In five cases gall-stones were found at operation; in three they were 'silent', and in the other two the attacks of colic were slight and were additional to the other symptoms, which were of much longer duration. The importance of prolapse of the ascending colon as an etiological factor in pathological conditions of the gall-bladder and ducts will be more fully discussed later.

SECONDARY OR END-RESULTS.

The clinical conditions I have described may remain stationary for years, but as a rule tend to be progressive, and the original symptoms may be replaced by others, which may be called 'secondary' or 'end-results'.

1. **Chronic Duodenal Ileus** (11 cases).—Although, as has already been pointed out, this condition may be due to other causes, it is most commonly found in association with prolapse of the ascending colon and cæcum, being caused either by intermittent compression of the third part of the duodenum by the superior mesenteric vessels, or by duodenal inhibition, the result of sympathetic stimulation due to increased tension in the mesentery. It is, in these cases at least, not a separate clinical entity but a secondary or end-result of the prolapse of the ascending colon. When the 'bilious attacks', which are so frequent and severe in the duodenal type of case, become so overwhelming as to overshadow the other symptoms, and when the duodenal dilatation is extreme, it becomes necessary to describe the case as one of chronic duodenal ileus. The 'bilious attacks' begin usually with severe headache, nausea, and discomfort in the upper abdomen, and culminate in 'bilious vomiting'. The attacks cannot be aborted, and no relief is obtained until vomiting ensues. Although these patients are as a rule constipated, diarrhœa may follow an attack. It has already been mentioned that in this series of cases moderate dilatation of the duodenum was found in 53 and marked dilatation in 31. In 13 of the latter the dilatation was 'very marked', and in 5 of these so extreme that the decision to perform a colopexy and not a duodenojejunostomy was made with considerable hesitation. These 13 cases should therefore be classified as chronic duodenal ileus, but the number is reduced to 11, as in one ulceration of the lesser curvature, and in another mucous colitis, had supervened.

2. **Gastric or Duodenal Ulceration** (25 cases).—During the six years covered by this investigation I have operated in 163 cases of chronic gastric or duodenal ulcers (not including perforations). A pyloroplasty was considered sufficient in some involving the pylorus alone; in those of the lesser curvature a partial resection was, as a rule, performed; and in the majority of the remainder a gastrojejunostomy was necessary. There were, however, 25 (2 gastric, 6 pyloric, and 17 duodenal) in which a colopexy was performed. In more than half of these there was a definite history of symptoms which seemed to be due to prolapse of the ascending colon, but which, after a varying period, were replaced by symptoms of ulceration. In none was there a

history of any primary focus of infection in the abdomen, and in none was a focus found at the operation. In 5 of the 17 duodenal ulcers the strain of the loaded ascending colon falling directly on the duodenal wall seemed to be an important factor in the production of the ulcer, as in each case there was a developmental membrane attached to the wall at the site of ulceration. In one of these there was marked dilatation of the second part of the duodenum; and in three others marked dilatation of the first three parts from the drag on the superior mesenteric vessels. The importance of prolapse of the ascending colon as a factor in the causation of ulceration, and the degree of success which followed this method of treatment, will be dealt with later.

3. **Mucous Colitis** (17 cases).—Mucus is so frequently found in the stools, especially in the right iliac fossa type with marked constipation, that it may seem unnecessary to classify any of these cases separately, but the colon may be, in whole or in part, so affected with a mucous colitis that the original symptoms may be replaced by those of this distressing condition. That mucous colitis may originate in two entirely different ways seems clear: it may be the result of primary bacterial infection from within, or it may be secondary to stasis. It is with this latter type, associated with abnormal mobility and prolapse, that I am dealing. The difficulty of differentiating between them will be referred to when giving clinical results.

IV. THE RELATIONSHIP WITH CERTAIN FORMS OF APPENDICITIS.

As has already been pointed out when describing peri-appendicular developmental membranes, the wide-mouthed 'infantile' type of appendix is often found associated with a mobile dilated cæcum, and one might even affirm that abnormal mobility of the cæcum invariably accompanies the occurrence of this type of appendix. Cæcal stagnation may thus readily extend to the appendix, and therefore, without any obstruction of its lumen from within or without, there may be true colic of the appendix due to its attempt to evacuate fæcal contents, which occasionally take the form of coproliths. This condition of 'choked appendix', in which there is no actual stenosis of the lumen is, in my experience, very rarely seen except in association with abnormal mobility and dilatation of the cæcum. As already emphasized, the appendicular colic which may thus occur in patients who exhibit symptoms due to prolapse of the ascending colon and cæcum must not be confused with the characteristic pain of the right iliac fossa type of case. It is an additional condition, secondary to the other, just as biliary colic is.

Any surgeon who has had much experience of cases of acute appendicitis must have been impressed with the fact that in those cases where there is gangrene *en masse*, with thrombosis of the vessels, or where there has been intra-appendicular suppuration due to stenosis of the lumen, the cæcum usually, if not invariably, cannot be lifted out of the abdomen. Thus, as it would be unwise to disturb the retrocæcal tissues, the appendix must be removed, often with great difficulty, from the depths of the wound. On the other hand, he must also have operated on many cases in which the cæcum and suppurating appendix were easily delivered and the latter removed with ease. In these cases, although the appendix was full of pus, the onset of the

illness was not nearly so acute, nor the progress so rapid, as in the former type. The reason is that in these cases the appendix was originally wide-mouthed, the patient may have had repeated attacks of appendicular colic due to choking of the lumen with fæces, and there may even have been former attacks of actual suppuration with successful evacuation of the pus into the cæcum. The mouth has now, however, become blocked and the lumen is full of pus: there may be an ulcerated or gangrenous area opposite a coprolith, usually in the distal half. This type of suppurative appendicitis is invariably accompanied by abnormal mobility of the cæcum, and might be considered as one of the secondary or end-results of that condition.

Recurrent attacks of subacute inflammation in which the process stops short of suppuration may occur in cases unassociated with dilatation of the cæcum, in which there is actual stenosis of the lumen; but this must be very rare, as, owing to the closure of the mouth, suppuration would almost certainly ensue. On the other hand, there must often be an extension of inflammation from the 'cæcal sink' down through the wide mouth of the infantile appendix, and cessation of the inflammation which may take place is due to the ease with which the contents can be evacuated.

Obliterative changes are sometimes seen in appendices which have been the site of former suppurative or subacute attacks of inflammation, and are usually most marked at the tip. To this condition, which is secondary to abnormal mobility and dilatation of the cæcum, the term 'chronic appendicitis' may be applied, but to no other.

V. RELATIONSHIP WITH GASTRIC AND DUODENAL ULCERATION AND WITH GALL-BLADDER CONDITIONS.

GASTRIC AND DUODENAL ULCERATION.

There has been, and indeed still is, considerable diversity of opinion regarding the relative frequency of chronic gastric and duodenal ulcers. Rovsing[48] has reported that of 380 cases treated by him between 1898 and 1917, 222 were gastric and only 158 duodenal (a proportion of 1 to 0·7). Of the 163 cases (not including perforations) operated on by me between 1920 and 1926, 70 were considered to be gastric and 93 duodenal (a proportion of 1 to 1·3). Of 718 cases operated on by Moynihan[49] between 1913 and 1922, 164 were gastric and 531 duodenal (a proportion of 1 to 3·2); and in the Mayo Clinic[50], of 5723 cases, 1191 were gastric and 4532 duodenal (a proportion of 1 to 3·8). Wilkie,[51] drawing attention to the frequency of the co-existence of both types, refers to 300 cases, of which only 37 were gastric, 221 duodenal (a proportion of 1 to 6), and 42 in which both were present. There should be little difficulty in determining the anatomical position of the ulcer except in those cases in which there has been a leakage. There seems to be no doubt that the ulcer is either gastric or duodenal, and that pyloric ulceration is rarely primary, being usually an extension from the duodenum. The diversity of opinion as to the relative frequency may be explained by some surgeons considering that ulcers involving the pylorus are gastric instead of duodenal in origin; but it is also, I think, due to the ease with which a gastric ulcer, especially if on the posterior wall, may be overlooked by both radiologist and surgeon. It is generally accepted that chronic gastric ulcers are usually single, and are most frequently found either along the lesser curvature or in the antrum pylori. Duodenal ulcers are often multiple and are almost invariably situated in the first segment, often within an inch of the pyloric ring, and most commonly on the anterior wall.

A great deal of work, experimental and clinical, has been done in connection with the mode of formation of gastric and duodenal ulcers. Rovsing[52] refers to the experimental production by Panum in 1862 of a gastric ulcer by the injection of small wax balls into the circulation, and to the description by Godivier in 1869 of a duodenal ulcer caused by an embolism of the pancreaticoduodenal artery. He is convinced that "there is a process of embolism at the foundation of most chronic gastric ulcers", and explains the association of chlorosis and anæmia with ulceration by the possible fatty degeneration of small vessels, with subsequent thrombosis. A vascular explanation has also been suggested by Wilkie[53], who thinks that emboli from omental veins in which thrombosis has taken place may be carried to the stomach or duodenum. Various forms of trauma—mechanical, thermal, and chemical—have been blamed. There can be no doubt, however, that a bacterial infection is the primary cause of many of the ulcers. It is well established that acute gastric ulcers may be infective in origin, due to infection of the mucous membrane by organisms or their products. The occurrence of acute ulcers in infective diseases—for example, puerperal fever—is well known. It is also generally accepted that the majority of chronic ulcers of the stomach and duodenum arise out of acute ulcers which failing to heal have resulted in local necrosis, and that in this process the action of the gastric juice, either normal or hyperacid, plays a very important part. It has until recently been supposed that the organisms, from whatever source, reached their destination in the mucous membrane either directly or through the blood-stream, and Ogilvie[54] insists that organisms, the result of alimentary infection, reach the blood-stream by being absorbed by the gastro-intestinal mucous membrane, chiefly at aggregations of lymphoid tissue. Braithwaite,[55] however, has demonstrated (in my opinion conclusively) the probability of a lymphatic route (*Fig.* 399).

The normal flow of lymph from the terminal six inches of ileum, the appendix, and the cæcum is by vessels which unite to form a group of five or six efferents which pass to the chain of glands dotted along the line of the ileocolic artery. The main trunks then pass up in front of the third segment of the duodenum into the glands grouped round the origin of the superior mesenteric artery, and from there into the receptaculum chyli. The lymph-flow from the ileocæcal angle would normally not reach either the duodenum or stomach. Braithwaite found that in living animals, even when there is no obvious disease in any of the glands, indigo-carmine injected into the wall of the cæcum travelled up to the pancreas, and over it into the subpyloric lymph glands (a group situated at the bifurcation of the gastroduodenal artery at the angle between the first and second segments of the duodenum). Again, active tuberculous gland tissue implanted into the submucous layer of the cæcum in a cat resulted in tuberculous infection of glands from the ileocæcal angle up to a gland in the subpyloric group. In the living human subject it was found possible to get the dye to pass from the appendix wall, through the ileocolic chain, to the superior mesenteric group, but also to the subpyloric group.

Although normally the flow of lymph is from the periphery of each great lymph-shed to its termination in one or other of the large groups of glands,

Braithwaite is of the opinion that "there are in existence reserve sets of lymph channels as well as lymph glands which come into play under unusual pressure of lymph-flow or in the presence of obstruction in the normal channels due to disease". Thus, if there is a chronic lymphadenitis or lymphangitis, the lymph-flow may be diverted from the superior mesenteric glands to the

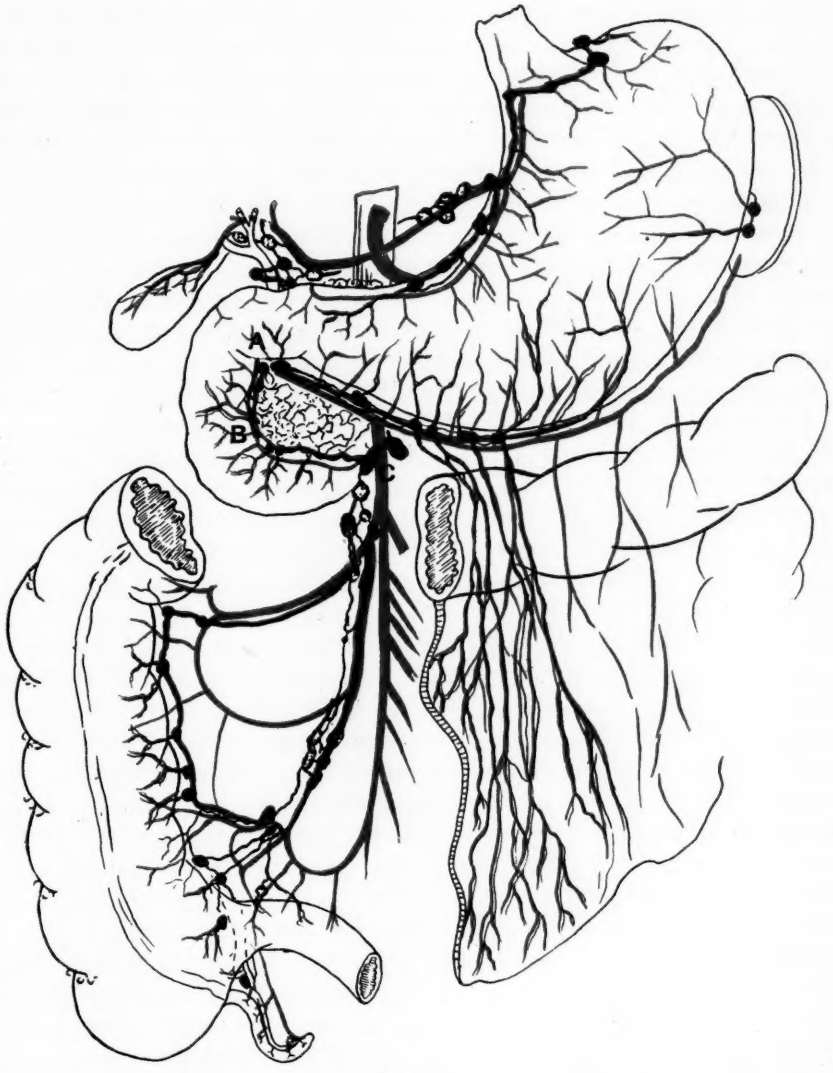


FIG. 399.—Diagram showing the visceral lymphatic glands and vessels. Note especially the pancreaticoduodenal vessels and glands (B) which connect the superior mesenteric group (C) with the subpyloric set of the hepatic group (A). (*After Braithwaite.*)

right over the head of the pancreas to the anterior and posterior pancreaticoduodenal glands (which lie along the anastomotic arch formed by the superior and inferior pancreaticoduodenal arteries in the concavity of the duodenum). From these glands the flow is to the subpyloric group, and thence through

the hepatic glands to the cœliac group. Now, if there is obstruction to the normal flow, it is easy to understand how it may from the pancreaticoduodenal glands reach the mucous membrane of the duodenum, from the subpyloric glands reach the mucous membrane of the lower part of the pylorus, and from the hepatic glands reach the upper part of the pylorus. Again, the normal flow from the mucous membrane of the lesser curvature of the stomach is through the superior gastric glands (which lie along the coronary artery) to the cœliac group. As these also receive efferents from the hepatic glands, it is easy to understand how by retrograde flow infective lymph may reach the mucous membrane of the lesser curvature from the hepatic glands. Again, the normal lymph-flow from the great omentum is to the inferior gastric glands (which lie along the right half of the greater curvature of the stomach), and thence to the subpyloric group. This was demonstrated by Braithwaite, who, laying gauze soaked in the dye on the lower free border of the omentum, found that the dye rose along the blood-vessels of the omentum and filled the inferior gastric glands. If, however, there is lymphatic obstruction, lymph may readily be diverted and, by retrograde flow, reach the mucous membrane of the greater curvature of the stomach.

The mucous membrane of the duodenum and stomach may thus be the target for infective material not only from the ileocæcal angle but from any part of the abdominal cavity. It is important to remember that it is not necessary for the omentum to be actually adherent to the focus of infection. The important part played by the great omentum, 'that great drain-pipe', in the removal of the various forms of phagocytic cells present in the peritoneal exudate in cases of infection in any part of the cavity, has been previously described by me.[56]

The effect produced by the arrival of infective material in the mucous membrane of the duodenum and stomach depends, among other factors, on the virulence of the infective agent. Thus, in the case of the duodenum, should the infection be acute, there may be an acute duodenitis with a lymphatic spread to the gall-bladder and ducts. If, however, the infection is less acute, there occurs erosion of the lymph follicles of the duodenal wall. This one would expect to find most commonly in the place where there is the greatest collection of lymphoid tissue, especially as this corresponds with the 'anæmic spot' of Mayo, on the anterior wall about one inch beyond the pylorus, towards the upper or antimesenteric border. Again, as this area is more exposed to the action of the acid gastric secretion, erosions are less likely to heal than in the distal parts of the duodenum where the contents are alkaline. The well-established fact that chronic duodenal ulcers are almost invariably found in the first segment seems to support this theory. Similar conditions are found in the stomach, and, as in the case of the duodenum, one would expect those areas of the mucous membrane which are richest in lymphoid tissue to be most commonly affected. Bolton[57] states that the lymph follicles in the lesser curvature and near the pylorus are twice as numerous as, and larger than, in the rest of the stomach, and it is in these areas that gastric ulcers most commonly occur.

That pathological conditions of the appendix may be closely associated with the causation of gastric and duodenal ulceration has been long recognized

(Mahnert,[58] MacCarty and McGrath,[59] Paterson,[60] Burk,[61] Moynihan,[62] Sherren[63]). As already noted when discussing 'reflex dyspepsia', Moynihan believes that 'appendicular dyspepsia' due to inflammation of the appendix is accompanied by acute congestion of the mucous membrane of the antrum pylori. Other observers, finding no changes in the gastric mucous membrane, have regarded the condition as purely reflex; and Tyrrell Gray, while admitting that the focus of irritation may be an appendix actually inflamed, believes that similar symptoms may be caused by the " mutual mesenteric drag of a controlling appendix ". In spite of this want of unanimity there can, I think, be no doubt that a flow of infected lymph, not only from the ileocæcal angle but from any part of the abdominal cavity, may be an important etiological factor in the production of gastric and duodenal ulceration, and that in a certain proportion the secondary result may be cured by the removal of the primary cause.

Wilkie, in describing the condition of chronic duodenal ileus, points out that duodenal stasis, in addition to causing toxic phenomena, may, " by allowing bacteria to multiply, be a source of infection to its own wall and to adjacent viscera which communicate with it ". Considering that the association of duodenal ulceration with chronic duodenal ileus is too frequent to be mere coincidence, he holds that duodenal stasis must be one of the predisposing causes of duodenal ulceration, and that the persistence of the stasis may explain the occasional failure of the usual operative treatment of that condition. While I am in agreement with this opinion, I have not found the association as frequent as Wilkie suggests. Of the 17 duodenal ulcers included in this series of colopexies, in 9 the duodenum was normal, in 5 it was moderately dilated, and in only 3 was there marked dilatation, in none extreme enough to merit the description 'chronic duodenal ileus'. Again, there was no ulceration of the duodenum in 24 of the 26 cases in which there was marked dilatation due to the drag on the superior mesenteric vessels, and of the 13 cases of chronic duodenal ileus there was ulceration of the lesser curvature of the stomach in 2, but none of the duodenum.

Waugh[64] is of opinion that the gastric and duodenal ulcers are relatively rare, but that when present they are caused by the strain of the loaded prolapsed ascending colon and cæcum falling directly on the duodenum, and by 'radiation' on the pylorus. He has been impressed by the fact that, while in all cases of ulceration the ascending colon was abnormally mobile, the stomach and duodenum had not prolapsed but had remained fixed in their normal anatomical position. Rovsing, also, has noted that ulcers developing in prolapsed stomachs are most commonly found " where a fold of the stomach has become fixed by peritoneal adhesions ", and attributes this to the " immovable crest of mucous membrane . . . especially exposed to traumatic abuse ". In the 17 cases of duodenal ulceration in this series the ulcer was in the first segment in all, on the anterior wall in 12, on the posterior wall in 3, and on both anterior and posterior walls in 2. In 5 of those on the anterior wall there was a definite developmental membrane, which could not be confused with an inflammatory adhesion, attached to and dragging on the duodenal wall at the site of the ulcer. In these cases the drag transmitted through the membrane seemed to be the important causal factor in the

production of the ulcer, especially as there was neither duodenal stasis nor appendicular infection present in any of them. (Four were cured by a right colopexy; the fifth, owing to involvement of the pylorus, required a gastrojejunostomy, performed at the same time.)

Waugh's purely mechanical theory of the causation of duodenal—and it may be also of gastric—ulcers may be sufficient to explain a few cases, but they are more often due to several factors. While in certain cases the chief cause may be either the direct transmission of the drag of the prolapsed ascending colon and cæcum, or the infective condition set up in the duodenum by stasis there, or the spread by aberrant or retrograde lymph-flow of infective material from the ileocæcal or other region of the peritoneal cavity, in the majority of cases all these conditions are present and take part in the formation of the ulcer.

The theory that abnormal mobility and prolapse of the ascending colon and cæcum invariably accompany gastric and duodenal ulceration is, however, not generally accepted. Morley[65] states that in cases of gastric or duodenal ulcers he is often unable to expose the appendix through the ordinary upper right rectus incision owing to normal fixation of the ascending colon. He admits, however, " that in some way a mobile proximal colon does predispose to gastric and duodenal ulceration ". Walton holds that it is relatively uncommon to find the two conditions associated, and supports his opinion by referring to the greater frequency of ulceration in males than in females. For various reasons the solution of this clinical problem is very difficult. An ulcer may be overlooked when abnormal mobility and prolapse of the ascending colon is being corrected. Much more readily may one fail to recognize abnormal mobility of the ascending colon when operating for ulceration. The incision does not usually permit of thorough examination of the lower abdomen, and, seeing no abnormal mobility of the duodenum or hepatic flexure, one is apt to presume that there is normal fixation of the whole of the ascending colon. Again, though theoretically advisable to remove or examine the appendix, it is in practice not always possible. No conclusion of value can therefore be based on the percentage of cases of ulceration in which the appendix was removed through a supra-umbilical incision. Thus, although in 68 per cent of the cases of gastric or duodenal ulceration on which I have operated since 1920 I have noted abnormal mobility and prolapse of part or the whole of the ascending colon, in only 34 per cent have I removed the appendix.

I am convinced not only that some degree of abnormal mobility and prolapse of the ascending colon and cæcum is present in the great majority of cases of gastric and duodenal ulceration, but also that, accounting as it does for the direct drag, the duodenal dilatation and stasis, and the possible focus of infection in the ileocæcal region, it is in many cases the primary etiological factor. Wilkie is of the opinion that failure to recognize and correct a condition of marked duodenal dilatation is one of the causes for disappointment after the performance of a gastrojejunostomy for gastric or duodenal ulceration. I would go further and suggest that the disappointing result may be due to failure to recognize and correct the abnormal mobility and prolapse of the ascending colon which accounts for the duodenal dilatation.

GALL-BLADDER DISEASE.

Although many may refuse to accept abnormal mobility and prolapse of the ascending colon as the primary factor in the production of gastric and duodenal ulceration, its importance as a factor in the causation of various gall-bladder conditions is now generally accepted. I have already referred to the frequent occurrence of a gall-bladder ventral membrane in cases of visceroptosis. It was present in 56 of my cases, being confined to the gall-bladder and cystic duct in 26, but extending to the pylorus, duodenum, gastrocolic omentum, transverse colon, or hepatic flexure in 30. While this membrane may cause no interference, the drag of an abnormally mobile and prolapsed ascending colon may be transmitted through it to the gall-bladder and result in marked kinking at the junction of the gall-bladder and cystic duct. I have no doubt that, as in the case of the appendix, most of the so-called 'adhesions' which are seen in the vicinity of the gall-bladder are developmental and not inflammatory in origin, and that their division, without correction at the same time of the position of the colon, is not as a rule sufficient.

Stagnation of the contents of the gall-bladder may, however, occur in cases of visceroptosis without any mechanical obstruction. Every surgeon has repeatedly noted with some anxiety, when operating for acute appendicitis, that the gall-bladder was tense and difficult to empty, although there was no obvious obstruction or kinking. While this condition may be a co-existent suppuration in the gall-bladder and appendix (hence the anxiety), usually it is merely stasis due to active reflex inhibition, the focus of irritation being the inflamed appendix. So also in cases of visceroptosis, distention of the gall-bladder may be due to reflex inhibition from abnormal mesenteric tension elsewhere. I have already referred to cases, Waugh's 'biliary type', in which, although attacks similar to biliary colic had been experienced, no calculi were found at operation. This condition of 'Stauungsblase', referred to by Aschoff,[66] Wilkie,[67] and others, may thus be due either to mechanical kinking or to reflex inhibition, in both cases being associated with right-sided ptosis. Reference has already been made to so-called 'silent' gall-stones as one of the causes of reflex dyspepsia. It may be that in many of these cases right-sided ptosis is the original cause both of the biliary stasis, which preceded the formation of the calculi, and of the reflex dyspepsia.

Interference with the evacuation of the contents of the gall-bladder resulting in stagnation is undoubtedly a predisposing cause of gall-stones. Macrae,[68] while mentioning undue viscidity of the bile as a possible cause of stasis, points out as noteworthy the fact that "the majority of the usually accepted predisposing causes of gall-stones are all such as tend to exaggerate any ptosis which may be present". In common with Macrae I have noted in every case of gall-stones on which I have operated since 1920, and in which I have had an opportunity of examining the ascending colon, that this was found to be prolapsed.

As infective conditions of the gall-bladder and ducts play an important part in the formation of calculi, it is necessary to discuss the source and route of infection. Various channels of infection have been suggested. Wilkie, in

stating that "the adjacent viscera which communicate with it" are liable to spread of infection from a duodenum in which there is stasis, seems to imply that the spread is by the lumen of the ducts. Many accept the blood-stream as the usual route, but Braithwaite seems to have proved conclusively the probability of a lymphatic route. Normally the lymph-flow from the gall-bladder passes through the cystic lymph-gland to the hepatic glands. Most of the lymphatic vessels from the bile-duct pass directly to the lower hepatic glands, but those from the terminal portion of the common duct pass to the upper pancreaticoduodenal set. By retrograde flow infective material may easily be carried from these glands to the gall-bladder and ducts. The source of infection need not necessarily be situated in the immediate vicinity—for example, the duodenum—but may be in the ileo-cæcal angle or indeed in any part of the abdominal cavity. We have thus a logical explanation of the frequent association of gall-bladder stasis, with or without infection or calculus formation, with visceroptosis, and of the co-existence of suppurative cholecystitis and appendicitis.

VI. TREATMENT.

The treatment should be preventive or curative, the latter including non-operative and operative measures.

PREVENTIVE AND NON-OPERATIVE TREATMENT.

Preventive treatment should be directed chiefly against the possible exaggeration of congenital ptosis. Thus every effort should be made to prevent the occurrence of a faulty body formation of either the virginal or maternal type. The former, which is almost always an acquired not a congenital condition, may be avoided by good feeding, suitable physical exercises, drill, etc.; the latter by paying more attention than is customary to the condition of the abdominal muscles after pregnancy. At the same time indiscretions in diet and carelessness in evacuation of the bowels, which might impose an extra strain on the ptosed organs, should be guarded against.

Curative treatment is called for if preventive treatment has failed. If ptosis is well developed, and if, the balance being disturbed, stasis has ensued, an attempt should be made to restore the balance by removing the stasis. (As has already been pointed out, the onset of symptoms synchronizes with the onset of stasis.) The meals, which should be small and frequent, must be taken 'dry', and should invariably be followed by a short rest. Constipation should be combated by mild laxatives, not by violent irritating purgatives. The condition of the abdominal muscles may be improved by massage and suitable physical exercises. Rest in bed, especially if the foot of the bed is raised, may help in this attempt to get rid of the stasis by relieving the mesenteric strain, but this can have no effect in the correction of the ptosis, which becomes evident as soon as the patient again assumes the erect position. The wearing of a Curtis belt, strongly advocated by Jordan,[69] if well fitted and carefully applied, often gives relief; but this is merely temporary, and the popular but unintelligent use of abdominal supports has so many disadvantages that it ought to be condemned. I am convinced that the relief

obtained is due simply to the increased abdominal pressure and not to any upward displacement of the viscera. Not only have I noted, with the opaque meal, the failure of a Curtis belt to alter the position of the stomach, but I have several times, on opening the abdomen, been convinced that the belt which had been worn must have been pushing the ascending colon and cæcum farther down into the pelvis. Gastric lavage is, of course, useless, as delay in gastric evacuation is secondary to stasis in the ascending colon, and to correct, even if that were possible, the secondary result without relieving the primary condition would be useless. Colonic lavage also is unsatisfactory, and usually of little use, as, in the early stages at least, the stasis is most marked in the cæcum and ascending colon.

This non-operative attempt to cure the stasis, and thus restore the balance, should always be made and may succeed. It will, however, probably fail if there is a marked degree of faulty body formation, if there are developmental membranes actually causing kinking, or if the symptoms, especially the toxic phenomena, are well developed.

OPERATIVE TREATMENT.

If the non-operative attempt fails, operative treatment is called for, but it must be distinctly understood that this is to be directed against ptosis and not stasis; if the former is corrected, the latter will cure itself, provided that gross secondary changes have not supervened. If the condition of stasis has been correctly explained by the stimulation of the mesenteric inhibitory nerves by mechanical traction of the ptosed viscus, removal of the traction by normal fixation of the offending viscus would abolish the inhibition and would thus cure the stasis.

Abnormal mobility is most commonly found in the cæcum and ascending colon, the last parts of the large bowel to be immobilized. The transverse colon may also be abnormally low, but ptosis of this part of the bowel and of the other viscera—the stomach, duodenum, and right kidney—is usually secondary to ptosis of the ascending colon. The attempt to cure the condition should as a rule be confined to the ascending colon and cæcum. Fixation of the cæcum alone (cæcopexy) may be sufficient in some cases, provided that the hepatic flexure and the upper part of the ascending colon are normal. In others, however, it may be necessary, in addition to fixing the ascending colon (right colopexy), to suspend the proximal part of the transverse colon.

Since Wilms'[70, 71, 72] advocation of cæcopexy for the conditions of cæcum mobile, various operations for the fixation of the cæcum and ascending colon have been practised. I have employed the simple but effective method described by Waugh. (*See Figs.* 400–403.) The appendix, if present, having been removed, the mobile ascending colon is drawn to the left and an incision is made along the 'white line' which marks the junction of the true and false mesenteries, from the hepatic flexure to the pelvic brim. An extraperitoneal space is thus formed, the floor of which consists of the iliopsoas and quadratus lumborum muscles, the latter crossed obliquely by the ilio-hypogastric, ilio-inguinal, and external cutaneous nerves, with the ureter lying along the inner side and the lower pole of the kidney exposed at the upper end. Into

this bed the ascending colon is rolled, and fixed by stitching the anterior longitudinal band to the cut edge of the false mesentery. I do not remove the fat and cellular tissue of the extraperitoneal space, as advocated by Waugh, but I pay particular attention to two points: the cæcum must be very carefully stitched and firmly cradled, and on reaching the hepatic flexure the suture line must deviate outwards from the anterior longitudinal band in order to avoid axial rotation of the bowel. An abnormally long prolapsed bowel may be safely tucked into a bed less than half its own length. I have been impressed with the fact that, while effective immobilization is required,

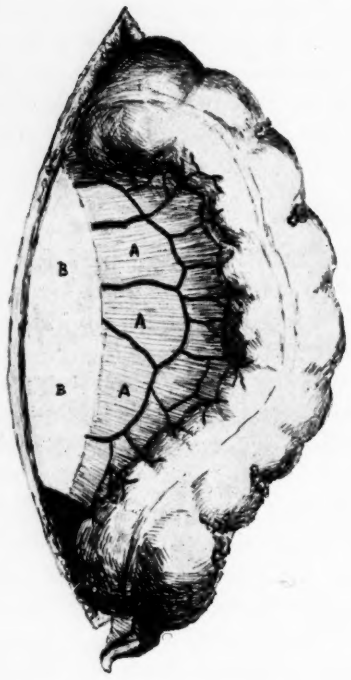


FIG. 400.—The abnormally mobile ascending colon and cæcum have been lifted out of the abdomen, revealing the true mesentery (A) with its arches of blood-vessels and the opaque, non-vascular false mesentery (B). (*After Waugh.*)

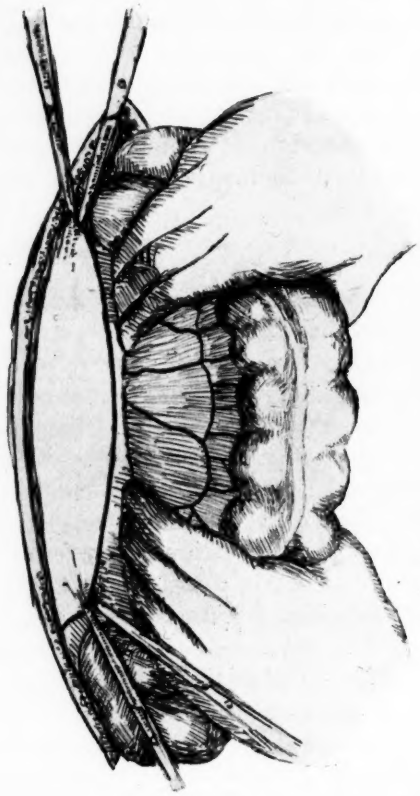


FIG. 401.—Showing the line of incision along the junction of the true and false mesenteries. (*After Waugh.*)

the greatly prolapsed bowel need not necessarily be fixed in its ideal anatomical position. I do not apply any antiseptic to the abdominal wall, as advocated by Waugh, as, since making a practice of changing my gloves and discarding needles used when stitching the bowel before closing the abdominal wall, I have had no case of wound infection.

Division of developmental membranes may also be necessary. As a Lane's membrane may cause traction on the terminal ileum after, if not before, replacement of the prolapsed cæcum, it should, if present, be divided. A Jackson's membrane may be ignored, but a right intercolic membrane

should be divided, as, by exaggerating the angulation at the hepatic flexure, it may aggravate the stasis in the ascending colon. Division of this membrane is indeed necessary in order to expose the anterior longitudinal band, with which it is fused, before fixation by Waugh's method can be performed. Membranes from the hepatic flexure or transverse colon to the right kidney, liver, duodenum, or pylorus should also be divided. Simple division of a gall-bladder ventral membrane and its extensions to the pylorus, duodenum, and gastrocolic omentum may be sufficient, but if biliary stagnation is marked it may be necessary to suspend or drain the viscus, having first removed gall-stones if present. Suspension or drainage of the gall-bladder, perhaps supplemented by shortening of the round ligament of the liver, may help to correct

FIG. 402.—The false mesentery has been turned outwards, exposing above the lower pole of the kidney, on the inner side the ureter, and, crossing the quadratus lumborum muscle, the ilio-hypogastric, ilio-inguinal, and external cutaneous nerves. (*After Waugh.*)

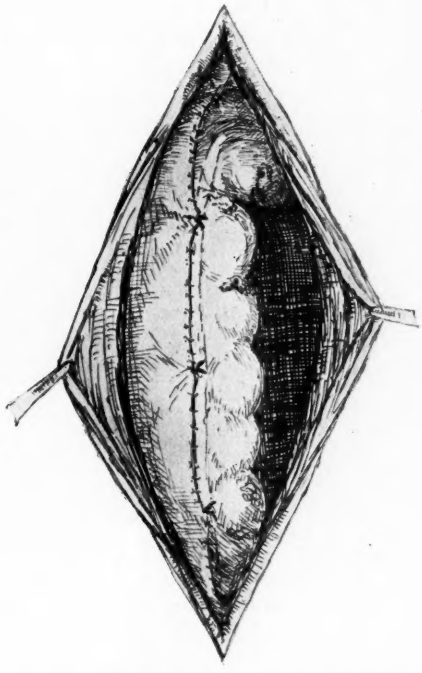


FIG. 403.—The ascending colon and cæcum having been rolled into the extra-peritoneal space, the cut edge of the false mesentery has been stitched to the anterior longitudinal band. (*After Waugh.*)

a degree of hepatosis. If possible the splenic and sigmoid flexures should also be inspected; a left intercolic membrane should be divided, but in very few cases have I thought it necessary to divide mesosigmoid membranes, although their presence was frequently noted.

As the ascending colon is primarily responsible for the abnormal mobility and prolapse of the right kidney, removal of the drag by fixation of the bowel is usually sufficient, the kidney soon receding to its normal level. In very few cases need a nephropexy be done in addition to a right colopexy, and the performance of the former without the latter is not only illogical in

theory but unsatisfactory in practice. In two cases in this series a nephropexy had been previously performed without benefit. In none of my cases was the kidney fixed at the same time as the colon, but in one the occurrence of a severe prolonged post-operative pyelitis necessitated a nephropexy two years later.

Very difficult problems must be faced if secondary results have supervened, as it is often extremely difficult to determine whether correction of the primary factor alone is sufficient or whether the secondary changes themselves demand additional treatment.

Thus, if the degree of *duodenal dilatation* present is so marked, and the symptoms of duodenal stasis are so profound, that the condition merits the description 'chronic duodenal ileus', the question of the advisability of performing a duodenojejunal anastomosis arises. It must be admitted that a condition of marked dilatation due to a definite irremovable mechanical obstruction can be relieved only in this way. It should not be forgotten, however, that chronic duodenal ileus is usually not a separate clinical entity but merely one of the advanced manifestations of a right-sided ptosis. While it may be doubted whether in these cases the dilatation is due to partial mechanical obstruction, or to inhibition of the duodenal wall the result of sympathetic stimulation due to increased mesenteric tension, it can usually be cured by relieving the downward drag by performing a right colopexy. A duodenojejunostomy should not be resorted to if there is a reasonable chance of relieving the condition by a simpler, more physiological method. Wilkie finds that even in those cases which are due to prolapse of the ascending colon the dilatation may be so extreme that no hope can be entertained of the duodenal wall recovering without anastomosis; but the decision that this is necessary should be made only after grave deliberation. My experience that the duodenum may be enormously dilated and yet be capable of recovery will be referred to later when describing the results of colopexy. Wilkie[73] reports that of 64 duodenojejunostomies he has been able to trace 56. Of these, 23 were cured and 11 much improved. It is important to note, however, that of the 36 in which there was obvious obstruction 18 were cured, but that of the 20 in which there was no evidence of obstruction only 5 were cured. It is therefore not surprising to find that he has decided to perform fewer duodenojejunal anastomoses in the future. McConnell and Hardman report 14 cases of chronic duodenal ileus, secondary to prolapse of the ascending colon, treated with very satisfactory results by colopexy. In all cases of duodenal dilatation, no matter how extreme, which in my opinion were due to prolapse of the ascending colon, I have refrained from direct interference with the duodenum and have contented myself with the performance of a right colopexy. That I have reason to be satisfied with this method of treatment will be seen from the clinical results detailed later.

Again, there may be considerable difficulty in deciding whether the presence of a *gastric* or *duodenal ulcer* demands additional or individual treatment. For a lesser curvature ulcer many surgeons are content with the performance of a gastrojejunostomy with about 80 per cent cures. Others combine this with excision of the ulcer, but if this should be extensive or adherent the only satisfactory method is either a sleeve resection or a partial

gastrectomy. In one of my cases where there was chronic duodenal ileus with dilatation of the pylorus an ulcer of the lesser curvature seemed to require additional treatment. As the patient's condition on the table after the performance of a right colopexy was causing anxiety, further interference was postponed. The perfect recovery which followed, and the continued absence of any symptoms three years later, showed that the sleeve resection which had been contemplated was unnecessary. On the other hand, in another case of chronic duodenal ileus a colopexy, while curing that condition, did not prevent the formation of a lesser curvature ulcer nine months later. In cases of ulceration involving the pylorus alone, if there is not much induration, a pyloroplasty is usually sufficient; but if there is marked thickening with stenosis a gastrojejunostomy is necessary. Although many surgeons claim that 90 per cent of cases of duodenal ulceration can be cured by a gastrojejunostomy, others give less favourable figures, and advocate in addition infolding of the ulcer, or excision if anterior, or transduodenal cauterization if posterior. Waugh has reported several cases of ulceration in the first and second segments of the duodenum which have been cured or very much improved by a right colopexy alone. I have no doubt that many duodenal ulcers can be cured in this way, and a cure is more probable if there is a definite dragging membrane attached to the site of the ulcer, or if chronic duodenal ileus is present, as both these factors can be removed by a colopexy. If the ulcer has encroached on the pylorus without causing any marked degree of induration, it is wise to combine a pyloroplasty with the colopexy. If, however, there is much thickening round the ulcer, even though it may not encroach on the pylorus, a gastrojejunostomy is called for, as the cicatrization accompanying the healing which may follow a colopexy would probably result in stenosis. In two cases in this series, in addition to performing a colopexy, I had to replace a gastrojejunostomy which had been performed elsewhere unnecessarily, as in neither was there any question of ulceration. While the performance of a gastrojejunostomy is one of the simplest abdominal operations, the replacement of the anastomosis, if this has been performed unnecessarily, is one of the most difficult and unsatisfactory. To perform a gastrojejunostomy simply because there have been so-called 'stomach symptoms' with a low splashing stomach is, of course, worse than useless: it is discreditable. No matter how low the stomach may be, no matter how long it may take to empty, there is no justification for the performance of this drastic deforming operation if the condition can be cured by simpler methods.

Should the original symptoms be replaced by those of a *mucous colitis*, a secondary result of stasis, it may be wise to perform an appendicostomy in addition to a colopexy. The operation of appendicostomy, advocated by Lockhart-Mummery,[74] was at one time a popular method of treatment for intractable cases of mucous colitis. A very rapid improvement usually followed, but in many cases this apparent cure was purely temporary, and the symptoms recurred soon after lavage was discontinued. In those cases in which a permanent cure resulted, the deciding factor may have been the preliminary fixation of the cæcum to the lateral abdominal wall which some surgeons practised. The condition of the bowel may, however, appear so

hopeless that colectomy seems to offer the only possibility of cure. The colon is not, however, a degenerate, useless, harmful organ: it has a definite part to play in intestinal digestion, and its removal would be justifiable only if it could be accomplished with a small mortality and be followed by a high proportion of cures. Although the few statistical results of either partial or total colectomy which have been published vary considerably, there can be no doubt that it carries with it a high mortality. There can also be no doubt that the after-results are not too satisfactory. It may be very difficult to estimate whether or not the bowel still retains the power of recovery. One must judge this not merely by the local condition (supplemented by X-ray demonstration of its motor function), but by taking into consideration also the age of the patient, the duration and severity of the symptoms, and the primary cause. Provided that the condition is not a primary one, but is secondary to inhibitory stasis associated with abnormal mobility and prolapse, the power of recovery is extraordinary. The improvement in the wall of the bowel after a colopexy which I have actually seen at subsequent operations has convinced me that in very few cases is removal of either a part or the whole of the colon necessary. Resection should certainly not be resorted to except in very hopeless cases with very advanced stasis in which all the coats of the bowel wall are so diseased that its neuromuscular mechanism is damaged beyond recovery.

Attempts to correct the abnormally low position of the stomach and transverse colon without correcting the faulty position of the viscus primarily responsible are both illogical and unnecessary, as has already been pointed out in the case of the right kidney.

The earlier operative measures were, however, usually directed towards the cure of stasis, not ptosis. Simple division of developmental membranes, which may be causing kinking at the points of attachment or abnormal mesenteric tension, may relieve stasis for a time; but this method of treatment is as a rule quite ineffective, as the exaggeration of ptosis that necessarily follows is accompanied by aggravation of stasis. H. M. W. Gray and Anderson have advocated this method of dealing with a "mechanical disability . . . by mechanical means", and Jackson[75] claimed 75 per cent successes; but Walton[76] admits that the symptoms are prone to recur, and advises that it should be followed by medical treatment, massage, exercises, etc. Simple division is not sufficient: replacement of the prime offender, the ascending colon, is necessary; to enable this segment of the bowel to recover its normal neuromuscular condition, it must be placed in a more, not in a less, favourable position. Ileosigmoidostomy, ileoproctostomy, and cæcosigmoidostomy may relieve the colonic stasis for a time, but are not followed by a permanent cure, for the offending bowel, although short-circuited, is left in its faulty position.

Appendicectomy.—Removal of the appendix, either when it is normal or in cases where the appendicular colic due to its fæcal contents is merely additional to the characteristic symptoms of prolapse of the ascending colon and cæcum, is useless. There can be no doubt that the results of the removal of the so-called 'simple', 'quiet', or 'chronic appendix' are unsatisfactory, whereas if the appendix, and it alone, were at fault they should be almost

invariably good. Battle[77] reports that of 1000 cases of 'appendicitis' operated on in the quiescent period more than 50 per cent showed merely 'catarrhal changes'. Guillaume[78] also is impressed with the doubtful pathology of the condition usually described as 'chronic appendicitis', and with the frequent recurrence of the symptoms soon after the removal of the appendix. Lichty[79] reports that of 400 cases operated on for 'chronic appendicitis' only 225 were cured, the diagnosis being evidently wrong in 175. Hathaway[80] points out that in many cases of so-called 'chronic appendicitis' the recurrence of the original pain soon after the removal of the appendix is invariably attributed to post-operative adhesions. The question ought to be cleared up: if the results of removal of the appendix in cases of so-called 'chronic appendicitis' are bad, doubt will be produced in the lay mind about the necessity of operation even in acute cases. I have already discussed the relationship between certain forms of appendicitis and prolapse of the ascending colon and cæcum, and have explained in detail the only condition to which the term 'chronic appendicitis' should, in my opinion, be applied. I have also drawn attention to the possibility of mistaking peri-appendicular membranes of developmental origin for true inflammatory adhesions. The appendix may, of course, be actually deformed by these membranes and evacuation of its contents interfered with. This, perhaps combined with the tendency of this functionless organ to undergo obliterative changes, may give rise to inflammatory changes which result in the formation of true inflammatory adhesions. Many appendices, however, are removed with satisfaction at the time to the surgeon, because those membranes are considered to be the result of former attacks of 'catarrhal inflammation', but with no permanent benefit to the patient. It may be difficult to differentiate between developmental membranes and inflammatory adhesions which have arisen independently or in consequence of previously existing developmental membranes; but I have no doubt that the so-called 'adhesions' which are so often seen in the vicinity of the appendix, and which are considered to be justification for its removal, are in most cases not inflammatory but developmental. Again, abnormal mobility of the cæcum may not be recognized because the appendix may be acting as a 'suspensory ligament', holding an abnormally mobile cæcum out of the pelvis. Removal of such a 'controlling appendix' may relieve local intestinal stasis for a time, but, as in the case of simple division of the other developmental membranes, it is followed by prolapse and further stasis. On the other hand, however, in cases of reflex dyspepsia due to an inflamed appendix, removal of the appendix alone is not only justifiable but is usually followed by a permanent cure.

One can thus expect satisfaction from removal of the appendix alone only in those cases in which that organ alone is at fault. It is not good surgery to remove a non-suppurating appendix without at the same time immobilizing the cæcum and ascending colon if these are abnormally mobile and prolapsed. If for some particular reason, or through an error in judgement, one is operating through a gridiron incision, it may be wise to perform a cæcopexy, a complete colopexy being impossible. This may be followed by a cure; but if the upper part of the ascending colon or hepatic flexure is prolapsed, a complete colopexy may be necessary later. If, however,

there is actual suppuration, simple removal of the appendix may be all that can safely be done, owing to the danger of infecting a retroperitoneal space; the prolapsed condition of the colon will, however, demand correction at a later date. Direct fixation of the bowel to the lateral abdominal wall is a tempting compromise in these cases, but is not usually satisfactory.

VII. ANATOMICAL AND PATHOLOGICAL RESULTS OF COLOPEXY.

The results which follow the operation of colopexy have been proved not only by repeated opaque meal examinations in the majority of the cases, but also by direct observation of the viscera in as many as 16 in which for various reasons the abdomen was subsequently reopened by me. Improvement in the position of the stomach almost invariably took place, although in many cases it was delayed for a considerable time. A rapid improvement in its functional activity, easily demonstrable by opaque meal examination, was, however, the rule.

FIG. 404.—Case of Miss D. W., *before operation*, showing very low position of the stomach, in the erect position immediately p.c.; a quarter of the opaque meal was still in the stomach 5 hours p.c.

FIG. 405.—The same case *a year and a half after operation*, showing improvement in position and tone of the stomach, in the erect position immediately p.c.; evacuation was now complete in less than 5 hours.

Figs. 404 and 405 show the case of Miss D. W., age 28: 'gastric type'. She had had abdominal discomfort for sixteen years, with much 'indigestion' and flatulence; erratic pain soon after meals but not dependent on the kind of food; marked constipation; both kidneys easily palpable; menstruation too frequent and profuse; very severe headaches, with profound mental depression. There had been no improvement after dry diet, etc., for six months. Right colopexy was performed, and progress after operation was slow but uninterrupted. A year and a half later the appetite was good; there was no 'indigestion', no pain, no constipation; menstruation was normal, there were no headaches, and the mental condition was entirely changed, the patient being now alert and bright; neither of the kidneys was palpable. Five and a half years after operation she is 'very well', and has been at work without interruption for four and a half years.

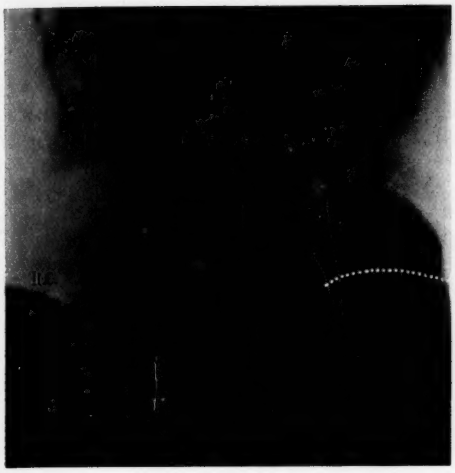


FIG. 406.—Case of Miss N., *before operation*, showing the stomach, in the erect position immediately p.c., the greater curvature being 5 inches below the interiliac line; a quarter of the opaque meal was still in the stomach 5 hours p.c.

FIG. 407.—The same case *one year after operation*, showing the stomach, in the erect position immediately p.c., the greater curvature now only 1 inch below the interiliac line; evacuation was now complete in less than 5 hours.

Figs. 406 and 407 illustrate the case of Miss N., age 32: 'duodenal type'. She had had 'stomach trouble', severe 'bilious attacks', and obstinate constipation for twenty years; constant 'indigestion', with very distressing flatulence and eructations several hours after food; loss of weight; right kidney very low. The general condition was very poor; she was disappointed, irritable, and emotional. There was no improvement after dry diet and a Curtis belt for one year. Right colopexy was done, and one year later all the symptoms were completely cured: the general nutrition was greatly improved, and the patient was 2 stones heavier; the right kidney was not palpable. Six years after operation she was 'perfectly well'.

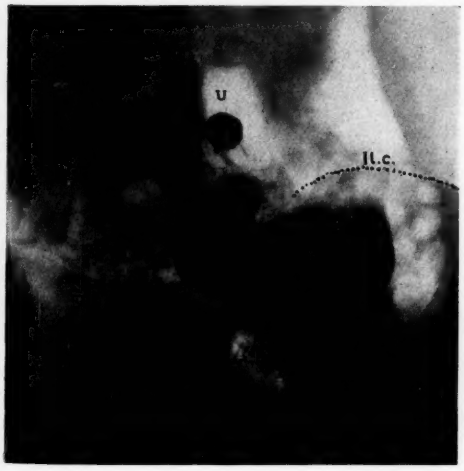


FIG. 408.—Case of Miss R. W., *before operation*, showing the stomach, in the erect position immediately p.c., hypotonic and low.

FIG. 409.—The same case *one year after operation*, showing improvement in position and tone of the stomach, in the erect position immediately p.c.

Figs. 408 and 409 are from the case of Miss R. W., age 22 : 'right iliac fossa type'. Patient had had abdominal discomfort for three years, with 'indigestion' and flatulence, erratic right-sided abdominal pain not connected with meals, and constipation. The right kidney was very low. The general condition was very poor. Right colopexy was performed; one year later all symptoms were cured; the general condition is greatly improved, and the patient is 1 stone heavier. The right kidney is not palpable.

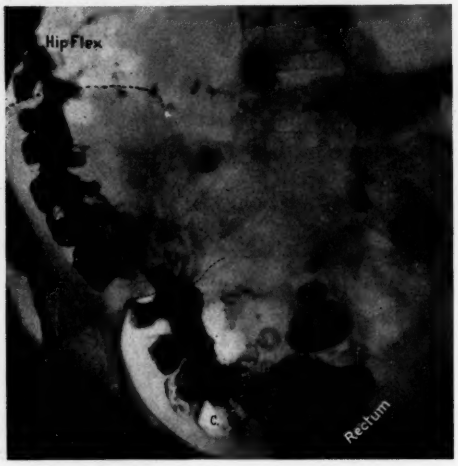


FIG. 410.—Case of Mrs. J., *before operation*, showing, in the supine position, prolapse of the ascending colon, the cæcum lying in the pelvis ; the barium reached the rectum in 24 hours.

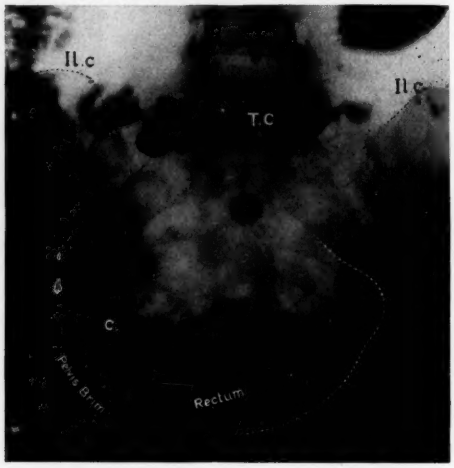


FIG. 411.—The same case *one and a half years after operation*, showing, in the supine position, the improved position of the ascending colon and cæcum ; the barium reached the rectum in 12 hours.

FIG. 412.—The same case, *before operation*, showing the stomach, in the erect position immediately p.c., hypotonic and very low ; only half empty 9 hours p.c.

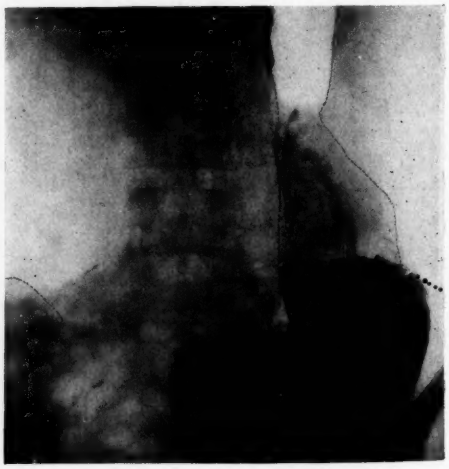


FIG. 413.—The same case, *one and a half years after operation*, showing improvement in the position and tone of the stomach, which now empties in 7 hours.

Figs. 410–414 are from the case of Mrs. J., age 34 : 'gastric type'. She had had abdominal pain for nineteen years, with constant 'indigestion', frequent vomiting and very distressing flatulence and eructation soon after food ; obstinate constipation, with much mucus in the stools ; marked loss of weight. The general condition

was very poor, and she was excitable, irritable, emotional, with fits of depression and threats of suicide. Both kidneys were easily palpable. The appendix had been removed elsewhere without benefit, and she had been considered a 'hopeless neurotic'. Right colopexy was performed; a year and a half later all symptoms had disappeared, and she was 'restored to health and happiness'; neither kidney was palpable; weight was increased by 1 stone. Six and a half years after operation she has remained 'perfectly well', with a 'splendid appetite', and is now 3½ stones heavier than before operation.

FIG. 414.—The same case, *three years after operation*, showing continued improvement; evacuation now complete in 4 hours.

In cases in which chronic duodenal dilatation had been noted at the operation, improvement in this condition, not being easily demonstrable by opaque meal examination, could be proved only in those cases in which a subsequent operation was performed. Of the 31 cases in which 'marked dilatation' had been noted, the duodenum was actually seen to be normal at a subsequent operation in 4, and of the remaining 27 only 2 were not cured. Failure in these two cases did not, however, seem to be due to persistence of the duodenal dilatation. In 5 of these 31 cases the dilatation was so extreme that the decision that a duodenojejunostomy was not necessary was made only after considerable hesitation; and yet in one the duodenum was seen to be normal at a subsequent operation nine months later, and in the other 4 the perfect cure which followed the colopexy showed that a duodenojejunostomy would have been unnecessary. I have thus direct evidence that very marked dilatation of the duodenum, if due to prolapse of the ascending colon, disappears after a colopexy.

In one case an ulcer situated on the anterior wall of the duodenum one inch beyond the pylorus was found to have entirely disappeared at a subsequent operation two years later. The duodenum also, which had been 'markedly dilated', was now normal.

The position of the ascending colon and cæcum was found, both by opaque meal examination and by direct observation, to be satisfactory, and the former mobility to be markedly restricted. In only one of my cases has there been any question of the ascending colon becoming mobile and prolapsed after a colopexy, and this unsatisfactory result did not come under my personal observation. I was told by the surgeon who performed the second operation, a gastrojejunostomy, that 'the colon was very movable', but from the skiagrams shown me I think that this description may have applied to the transverse and not to the ascending colon. In several cases it was noted at the second operation that the hepatic flexure was much less acute than before; this was repeatedly seen in opaque meal examinations. (*See Figs.* 415, 416.)

The improvement in the condition of the wall of the colon was very

striking. Increased rapidity in the passage of the opaque meal was noted in the majority of the cases, and in 6 the change from a thin-walled, much dilated bowel to an apparently normal bowel was actually seen at a subsequent operation. At several of the second operations, also, I noted that developmental membranes previously present had not re-formed after division.

The rapidity with which the prolapsed right kidney, no matter how low, recedes to its normal level is remarkable. In none of my cases was a nephropexy performed at the same time as the colopexy; but of the 132 cases in which the kidney was abnormally low, in only 16 could it be palpated at

FIG. 415.—Showing, *before operation*, in the erect position, prolapse of the cæcum and acute angulation at the hepatic flexure.

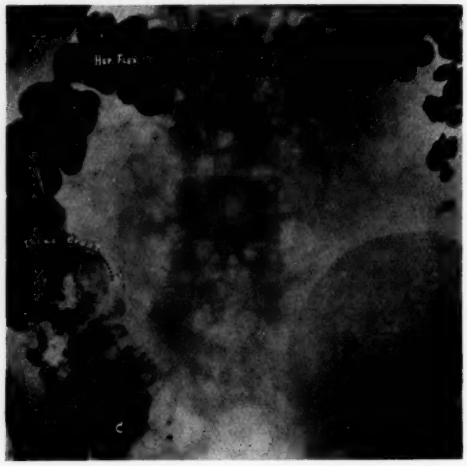


FIG 416.—The same case as *Fig.* 415, showing, *six months after operation*, in the erect position, improvement in the position of the cæcum and opening out of the hepatic flexure.

the end of six months, in 6 the lower pole alone could be felt after twelve months, in 2 no improvement at all resulted—the reason for failure in both being the occurrence of severe prolonged post-operative pyelitis. In none of the 27 cases in which the left kidney was abnormally low could it be palpated six months after the operation.

VIII. THE CLINICAL RESULTS OF COLOPEXY.

While this must not be considered an experimental study, I have, since 1920, deliberately agreed to operate on every patient brought to me whose condition was in my opinion primarily due to abnormal mobility and prolapse of the ascending colon, no matter how far advanced the secondary results were. Leaving out some 8 patients operated on in Bellahouston Pensions Hospital, with whom I have found it quite impossible to keep in touch, I am able to pass under review 242 consecutive cases in which a colopexy by Waugh's method was performed between March, 1920, and July, 1926.

In order to arrive at an absolutely accurate estimate of the value of this method of treatment I have personally kept detailed notes of every case, have seen the great majority of the patients at regular intervals, and have kept in touch with those who could not be seen by communicating with both patient and doctor. Thus, with only three exceptions, these cases have been traced up to date, and of the present condition of those who survive I have personal knowledge.

There have been many failures, and others have been so little improved that they are considered to be failures. One patient died suddenly on the fourteenth day after operation, and post-mortem examination showed that the cause of death was a large pulmonary embolism. Another developed acute intestinal obstruction on the sixth day after operation : a second laparotomy showed that this was caused by a fold of gauze which had been left in the abdomen, and notwithstanding a temporary improvement death ensued on the eleventh day. In another case, in which the mental symptoms overshadowed the others, the operation being followed by no improvement, the patient found a way out of her misery a year and a half later by cutting her throat. Another, after making an extraordinarily good recovery, developed acute phthisis one year after operation and died a few months later. This patient's mental depression before operation (partly due to an unsuccessful attempt elsewhere to cure her condition by removing her appendix) was so profound that she had attempted suicide. Four months after the colopexy her doctor reported that she was " making wonderful progress and was heavier than at any time within the past fifteen years ". As more failures occurred in cases in which secondary results had replaced the original symptoms, it will be more convenient to scrutinize the results in these groups before giving a general estimate.

Of the 11 cases of *chronic duodenal ileus*, 8 were completely cured, 2 were very much improved, and only 1 was a failure. In this last case a thorough examination revealed no ulcer, either gastric or duodenal—merely gastroptosis with very marked dilatation of the duodenum due to the drag of a much prolapsed ascending colon. Having performed a colopexy I made the note that " the extreme dilatation of the duodenum may necessitate a duodenojejunostomy later ". After nine months of apparently satisfactory progress the patient developed an acute lesser curvature ulcer, but when performing a gastrojejunostomy I found that the duodenal dilatation had entirely disappeared. The colopexy, while curing the chronic duodenal ileus, had failed to prevent the formation of the lesser curvature ulcer, or to cure it if its presence had been overlooked.

The group of 25 cases in which *gastric* or *duodenal ulceration* had supervened presented many difficulties, and thus included a large proportion of failures. One case illustrates only too well the difficult problem, already referred to, of how far the surgeon should rely on the radiologist's opinion if that differs from the clinical diagnosis. My opinion in this case that there was a pyloric ulcer was not supported by the opaque meal examination, and the very incomplete investigation which a precarious anæsthesia permitted failed to reveal any abnormality of the stomach or duodenum. Accepting the radiological finding, I contented myself with fixation of the markedly

prolapsed ascending colon. The patient died on the thirtieth day after several severe gastric hæmorrhages, and post-mortem examination showed that there was an ulcer on the posterior wall of the antrum pylori, with exposure of the pancreas. The gastrojejunostomy I had expected to perform might have saved the patient: the colopexy I performed certainly did not. Another interesting failure occurred in the case of a patient who had suffered from 'indigestion' for many years and had once had a severe hæmatemesis. No ulcer could be found at operation, and the condition of general visceroptosis, which was very marked, was apparently cured by a colopexy. After four years of perfect health there was a recurrence of severe hæmatemesis with a fatal result. The colopexy had failed to prevent the formation of an ulcer, or to cure it if its presence had been overlooked. In one case, as the duodenal ulceration by encroaching on the pylorus had resulted in stenosis, a gastrojejunostomy was performed at the same time as the colopexy. In 3 others a gastrojejunostomy was found to be necessary at a later date as the ulcer had not healed, or in healing had resulted in pyloric stenosis. In 3 others the failure was due not to persistence of the ulcer but to other conditions. Thus, in one case in which there had been 'indigestion' for more than ten years with marked constipation and much mucus in the stools, and with profound mental depression in addition to the usual toxic manifestations, an ulcer of the posterior wall of the pylorus was found and a colopexy performed. Although in her doctor's words "she was immensely benefited physically, her mental condition gradually became worse: she died of some form of leukæmia two years later". Two others, although not cured, were very much improved. The remaining 15 (1 lesser curvature, 2 pyloric, 12 duodenal) have remained absolutely cured in every respect for periods up to seven years after operation. Thus, although the results of this method of treating ulcers of the stomach and duodenum were not as successful as one would have wished, the fact that 15 out of 25 were cured by colopexy, supplemented in 4 cases by pyloroplasty, without having recourse to gastrojejunostomy, encourages me in the belief that if the prolapse of the ascending colon, which is undoubtedly an important factor in the production of the ulcer, is corrected, a cure may be expected in the majority of cases. Failure was due either to the ulceration being too active or too extensive, or to the other results of the prolapse being too profound.

The *mucous colitis* group of 17 cases, in which the original symptoms had been replaced by those of a mucous colitis, provided many disappointments. Some of these may have been due to failure to appreciate the primary cause of the condition. While in all the condition was considered to be secondary to abnormal mobility and prolapse of the ascending colon, it may be difficult in the late stages to differentiate between cases of this type and those in which the condition is due to primary bacterial infection from within. The chief cause of failure, however, was undoubtedly the fact that the condition was too far advanced, and that toxic manifestations, especially mental depression, were too well established. One woman, age 55, who after many years of ill health was in a most pitiable condition of physical and mental misery, and who was apparently making satisfactory progress, died, after

twenty-four hours' unconsciousness, three weeks after her operation. Post-mortem examination showed that the cause of death was cerebral œdema. Another woman, who had been a chronic invalid for twenty years, and who, having been obstinately constipated since childhood, had suffered from severe colitis for three years, showed no substantial improvement after operation. While another patient, whose three years' colitis was preceded by thirty-five years of marked constipation, insists that "the operation was worth while", as she feels so much stronger, has less discomfort, and more natural evacuations, her doctor expresses himself as disappointed with the result. She is accordingly considered a failure. Two others made rapid progress after operation, mucus disappeared from the stools within a few weeks, each added 1½ stones within six months, both remained very well for two years, and then both relapsed and are considered to be total failures. Of the 12 remaining, 5 although perfectly satisfied with their condition are considered 'very much improved' but not 'cured', as they have occasional flatulence and discomfort; but the other 7 are completely cured in every respect and have remained so for periods up to seven years. It is of interest to note that although in 5 of this group an appendicostomy was performed at the same time as the colopexy, only 1 of these was a complete cure. Of the remaining 12, 6 had already lost the appendix, and in the other 6 lavage was not considered necessary.

There are in addition 8 others whose improvement was so slight that they are considered to be failures, the number of which is thus brought up to 26, or slightly under 11 per cent.

Looking now on the other, more pleasing side of the picture, I find that of the 239 patients whom I have been able to trace, as many as 213 are satisfied with the result of the operation. (These do not, of course, include those on whom a gastrojejunostomy was performed, either at the same time or at a later date, as in spite of the cure obtained they are failures so far as a colopexy is concerned.) In the great majority of the cases the former symptoms disappeared within a few months of the operation. The appetite, which especially in the gastric type had usually been very fickle, rapidly improved, and patients often reported that they were at last able to eat 'anything and everything', or things they 'had not eaten for years' without regret. The relief from the distressing and often very embarrassing flatulence was also very greatly appreciated. The former 'bilious attacks', especially in the duodenal type, may persist for some months, but are much less severe and less frequent. This delay may be reasonably accounted for by the time taken for the duodenal dilatation to disappear. Although the characteristic pain, either after food or in the right iliac region, soon disappeared, there was often some degree of discomfort in the right side of the abdomen for some months. The recovery from constipation, no matter how obstinate it may have been, is one of the most satisfactory results. A degree of constipation may persist, but as a rule the condition is absolutely cured. Mucus soon disappeared from the stools even in cases of severe colitis. As might be expected from the rapidity with which the right kidney, if prolapsed, regained its normal position, the dragging pain formerly experienced in the right lumbar region soon disappeared. In only one of the renal type was a complete cure not

obtained. Another satisfactory result, which occurred too frequently to be mere coincidence, is the disappearance of menstrual irregularities. In more than half of the female cases in the series (185) the periods were either too frequent, irregular, or profuse, and in more than 80 per cent the cure which followed the colopexy included the disappearance of these symptoms. Of those previously married, 9 have had children since the operation, 5 for the first time; of 16 married since the operation, 9 have had children. (In no case did the pregnancy result in a recurrence of the former symptoms.) There was as a rule a rapid improvement in general nutrition, with steady increase in weight.

One of the most satisfactory results was the improvement in the abnormal mental condition, which was present in varying degree in 63 cases. In many of these there was merely 'nervousness', irritability, discontent, disappointment, or annoyance at the failure of previous treatment, but in others there was profound depression and apathy. While the mental outlook as a rule became rapidly brighter, it was not surprising to find that in the more severe forms the abnormality was exaggerated for a time immediately after the operation. In 3 cases in which the mental condition was primary, operation for the relief of secondary physical changes resulted in failure. On the other hand, in only 5 of the remainder did operation fail to cure both the physical and mental condition. The change in many cases was most striking, and the results which have given me most satisfaction, perhaps because least expected, have been in patients who had been condemned as 'hopeless neurotics', 'chronic abdomens', 'painful women', or even 'mental degenerates'. That the relief from the mental symptoms was permanent proves that it was not due merely to the suggestive effect of the operation.

Of the 213 patients who are satisfied with their present condition, 44 are in my opinion only 'very much improved', as they still have some abdominal discomfort, occasional flatulence, some degree of constipation, etc. The remaining 169 are literally 'cured', and the permanence of the cure may be judged from the number of years that have elapsed since the operation.

Table I.—Sex and Age Incidence.

	Under 10	10–19	20–29	30–39	40–49	50–59	60–69	Total
Female ..	1	19	63	57	33	11	1	185
Male ..	0	9	10	24	9	3	2	57
Total ..	1	28	73	81	42	14	3	242
Percentage	0·4	11·6	30·1	33·5	17·4	5·8	1·2	100

It is of interest to note that the proportion of females to males, almost $3\frac{1}{4}$ to 1, bears out the statement that a faulty body formation is much more common in the former than in the latter. The youngest, a female, was 5; the oldest, a male, was 64; 63·6 per cent were between 20 and 39.

Table II.—Results in 239 Cases (3 of the 242 could not be traced).

Results	Years since Operation							Total	Per-centage
	1–1½	1½–2½	2½–3½	3½–4½	4½–5½	5½–6½	6½–7½		
Cured	10	21	30	31	23	41	13	169	70·7
Very much improved..	5	7	13	5	6	5	3	44	18·4
Failures	0	4	5	5	4	3	5	26	10·9

Table III.—Results According to Type of Case.

Type of Case	Cured	Very much Improved	Failed	Total
Gastric type	27	9	5	41
Duodenal type	21	8	3	32
Right iliac fossa type	86	17	4	107
Renal type	5	1	0	6
With gastric or duodenal ulceration ..	15	2	8	25
With chronic duodenal ileus	8	2	1	11
With mucous colitis	7	5	5	17
	169	44	26	239

In order to show, as simply as possible, the results according to the degree of visceroptosis present, I have divided the cases into three categories. In those of the first degree the prolapse was limited to the ascending colon and cæcum; in those of the second degree the stomach and right kidney in addition were abnormally low; and in the third degree there was general visceroptosis.

Table IV.—Results according to Degree of Visceroptosis Present.

Degree of Ptosis	Cured	Very much Improved	Failures	Total
First degree	89 (82·4 per cent)	10 (9·3 per cent)	9 (8·3 per cent)	108
Second degree	44 (63·8 per cent)	19 (27·5 per cent)	6 (8·7 per cent)	69
Third degree	36 (58·1 per cent)	15 (24·2 per cent)	11 (17·7 per cent)	62

Conybeare and other writers have expressed the opinion that attempts to deal with individual organs in cases of general visceroptosis are useless. Wilkie,[81] trying to explain the diversity of surgical opinion on the efficacy of colopexy, suggests that those surgeons who are satisfied with their results probably operate only on those cases in which the prolapse is limited to the ascending colon, the other viscera being normal. He gives as his opinion

that while the operation may have some beneficial result in such instances, its practice in cases of general visceroptosis is " absolutely unsound in theory and never successful in practice ". As can be readily understood from the results detailed above, I do not agree with either the suggestion or the opinion. Although the percentage of cures diminishes as the extent of the visceroptosis increases, the fact that of 62 cases ot general visceroptosis 36 were absolutely cured, 15 were very much improved, and only 11 were failures, cannot be overlooked. This undoubtedly supports the view that ptosis of the other viscera is very frequently secondary to ptosis of the ascending colon, and that a considerable degree of success may be expected from attempts to cure the condition of general visceroptosis by fixation of the ascending colon. As already pointed out, a cure cannot be expected if gross secondary changes have supervened, as these may be too far advanced to be remedied by attention to the primary factor alone. This is borne out by the fact that in 8 of the 11 failures mentioned above secondary changes had taken place.

Table V.—Results in Third Degree of Ptosis.
(General Visceroptosis).

	Cured	Very much Improved	Failures
Gastric type	13	4	1
Duodenal type	11	3	2
Right iliac fossa type	2	2	0
Renal type	2	1	0
With gastric or duodenal ulceration ..	2	1	2
With chronic duodenal ileus ..	2	1	1
With mucous colitis	4	3	5
	36	15	11

Of the 239 patients, 51 had already undergone previous surgical operations without benefit (in 33 one, in 13 two, in 3 three, and in 2 as many as four and five operations). *Of these* 51 *a right colopexy cured all but* 8. That this result was not due simply to division of post-operative adhesions, as has been suggested, is shown by the fact that although these were present in 12 they were divided in only 3.

Again, only 6 of my patients have had, at other hands, later operations, and with one exception none had any reference to the condition for which the colopexy was performed. I have thus the assurance that, with this one possible exception, no other operative procedure has succeeded where my visceral fixation failed.

It seems therefore reasonable to assume that in at least the majority of the 213 cases the symptoms were due to abnormal mobility and prolapse of the ascending colon and cæcum, and that these patients owe their present condition of well-being to the operative correction of that abnormality.

The physician, who comes in contact with the surgeon's failures and not his successes, is naturally inclined to suggest that better results would be obtained if more discrimination were used in the choice of patient. That

cannot be denied. One would, of course, prefer to operate only on those who are not too old, who have had no previous unsuccessful operative experiences, who have developed no secondary results, whose mental condition is normal, and who are in a position to enjoy a prolonged and satisfactory convalescence. But what is to happen to the others? Can the psychotherapist expect to cure a secondary mental condition without correction of the primary physical one? Does 'general medical treatment'—a very vague expression—offer any hope of success after stasis is well established? In a recent communication Walton,[82] accepting the theory that pain in visceroptosis may be due to overaction of the sympathetic nerves, suggests that it might be relieved by their division. He does not, however, think that this is surgically practicable, owing to the difficulty of choosing the correct sympathetic nerves in the upper abdomen. It is disappointing to note that he makes no reference to the much simpler method of abolishing sympathetic overaction by relieving the abnormal mesenteric tension by visceral fixation, and that he feels that "relief should lie in the future . . . by the wider introduction of a healthier open-air life".

As the result of my investigation I am convinced that the majority of these difficult patients can be cured by the performance of a right colopexy, and that the earlier the attempt the more probable the cure.

IX. CONCLUSIONS.

In the evolution of the colon in man there is a gradual fixation by fusion and obliteration of the mesentery. Many variations, depending on irregularities in the disappearance of the mesentery, are found. The most common is abnormal mobility of the ascending colon (the last part to be immobilized) due to persistence, in whole or in part, of its primitive mesentery.

Abnormal developmental membranes are found associated with irregularities in the process of mesenteric fusion. These are due either to excess or to failure of normal physiological fusion. The method of formation and significance of each of the various membranes has been discussed in detail.

In the majority of cases of 'general visceroptosis' the primary condition is 'right-sided ptosis'. The strain of the loaded prolapsed ascending colon and cæcum has not merely a local effect but is transmitted to other viscera, both through the normal anatomical connecting links and through the developmental membranes. Ptosis of the right kidney, duodenum, and stomach is thus secondary to abnormal mobility and prolapse of the ascending colon and cæcum.

Stasis, the result of ptosis, cannot, however, be considered as a simple 'problem in mechanics'. Although purely mechanical kinking or traction may be an etiological factor in the production of stasis in the cæcum, ascending colon, gall-bladder, duodenum, and stomach, it is in many cases more reasonable to attribute the condition to inhibition due to sympathetic stimulation resulting from abnormal mesenteric tension either local or remote.

Visceroptosis may be unaccompanied by any symptoms; the onset of symptoms, which synchronizes with the onset of stasis, usually occurs when the congenital ptosis is exaggerated.

Great care must be exercised in deciding that the clinical condition in any particular case is due to visceroptosis and cannot be accounted for by other pathological conditions. Close co-operation and mutual confidence between surgeon and radiologist is necessary in order to reduce to as low a figure as possible the number of affirmative and negative radiological errors, and thus increase the value of the opaque meal examination.

A definite train of characteristic symptoms may be attributed to abnormal mobility and prolapse of the ascending colon and cæcum; and various clinical types, depending chiefly on the character of the pain, may be recognized.

If stasis in the ascending colon and cæcum is allowed to persist, various secondary results may supervene. It is generally admitted to be a factor of great importance in the production of gall-stones and other gall-bladder conditions. There is also good reason for believing that it is very frequently the primary factor in the causation of gastric and duodenal ulceration. Chronic duodenal ileus is usually not a separate clinical entity, but merely an advanced manifestation of right-sided ptosis. Mucous colitis is usually secondary to colonic stasis, and fæcal stagnation or suppuration in a wide-mouthed appendix to cæcal stasis.

Preventive treatment should be directed against the possible exaggeration of congenital ptosis by checking the occurrence of a faulty body formation of either the virginal or maternal type. If this fails, and if stasis has ensued, an attempt should be made to 'restore the balance' by non-operative methods. If this is not successful, operative treatment must be resorted to.

The earlier operations were directed towards the cure of stasis. Simple division of the membranes may relieve stasis, but the relief is merely temporary. The exaggeration of ptosis that necessarily follows is accompanied by aggravation of stasis. Short-circuiting the colon, again, may relieve stasis, but as the offending bowel is left in its faulty position no permanent cure results.

Operative treatment should aim at correcting the condition of ptosis; the stasis will then cure itself, provided that secondary changes are not too well established.

In the correction of the ptosis, only the ascending colon and cæcum need be dealt with. The right kidney and stomach will, without direct interference, regain their normal position and functional activity. The poor results which followed the operations of nephropexy and gastropexy were due to failure to recognize and to correct the primary factor—the prolapse of the ascending colon. In cases of 'choked' or 'controlling appendices' it is necessary to correct the position of the mobile cæcum, and failure to cure so-called 'chronic appendicitis' by removal of the appendix alone is due to this omission.

Provided that the secondary results already referred to are not too far advanced, these may be cured by correction of the primary lesion. Thus marked gastric and duodenal stasis may be cured by a right colopexy, without resorting to either a gastrojejunostomy or a duodenojejunostomy; gastric and duodenal ulceration, if not too active or extensive, may also be cured without either a partial gastrectomy or a gastrojejunostomy; a colectomy is unnecessary in cases of mucous colitis secondary to colonic stasis.

That fixation of the ascending colon and cæcum by Waugh's method is permanent, that it is followed by permanent improvement in the functional activity of the colon, duodenum, and stomach, and in the position of the right kidney and stomach, have been proved, I think, conclusively.

It has also been shown that, without any attempt being made to eliminate unsuitable cases, a large proportion of patients suffering from symptoms that are attributable to abnormal mobility and prolapse of the ascending colon and cæcum may be permanently cured by a right colopexy.

The abnormal mentality associated with visceroptosis may be primary, but is usually secondary, and in this case should not be considered a reason for refusing to operate, although it is undoubtedly one of the chief obstacles to satisfactory treatment.

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CANCER OF THE MALE URETHRA: A REPORT OF TWO CASES, WITH A SHORT SURVEY OF THE SUBJECT.

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That cancer of the male urethra is a very rare disease is shown by the fact that no specimen of it can be found in any of the large museums which we have visited, including those of the Royal Colleges of Surgeons in London and Edinburgh.

Judging from the literature, the disease usually occurs deep in the bulbous portion of the urethra, where it may arise in Cowper's glands or other glandular structures. In no case have we been able to find a record or specimen of cancer of the urethra which has originated in a stricture; one of the cases here recorded is therefore unique.

Fig. 417.—*Case* 1. Cancer of urethra. A, Site of new growth.

CASE REPORTS.

Case 1.—This is one of the more ordinary variety, arising without apparent cause on the bulb of the urethra and running a rapidly fatal course.

S. D., age 69, was admitted to the Bridgwater Hospital with acute retention of urine on Aug. 22, 1922. He was relieved by catheterization, but subsequently had a suprapubic cystotomy performed because of the difficulty in passing instruments by the urethra. He gave no history of gonorrhœa. He was admitted to the Bristol General Hospital on March 21, 1923, with a suprapubic fistula.

On Examination.—The patient was a rather thin but generally healthy old man. There was an old suprapubic fistula, in which was a drainage tube. In the perineum was felt a densely hard mass in the position of the bulb of the urethra, which terminated anteriorly in the root of the penis by an abrupt craggy edge. Both testicles were moderately enlarged and tender.

Operation.—On March 27 a portion of the perineal growth was excised and sent to Dr. Hadfield, the pathologist, who reported: "Transitional-celled carcinoma infiltrating the deep tissues, with evidence of rather rapid growth."

It was not considered that any radical operation could be done which would give a prospect of cure; but the patient, having a great deal of pain in the perineum, begged for operative relief, and therefore on May 8 the growth was removed. The scrotum was split and the whole penis was removed back to and including the triangular ligament. The specimen is shown in *Fig.* 417.

FIG. 418.—*Case* 1. Low-power view of centre of growth, showing masses of transitional epithelium, some showing central necrosis (N). (Obj. Leitz 3a, Oc. × 8.)

AFTER-HISTORY (from Dr. Thompson, of Bridgwater).—The patient was relieved of his pain, but in August, 1923, three months after the operation, a fungating growth appeared in the perineum and another quickly followed over the pubes. The patient's general condition slowly deteriorated, and he died in March, 1924.

DESCRIPTION OF SPECIMEN AND SECTIONS.—On slitting open the urethra from in front and cutting away the anterior portion of the growth, the latter is seen to be a dense mass about $3\frac{1}{2}$ in. long and $1\frac{1}{4}$ in.

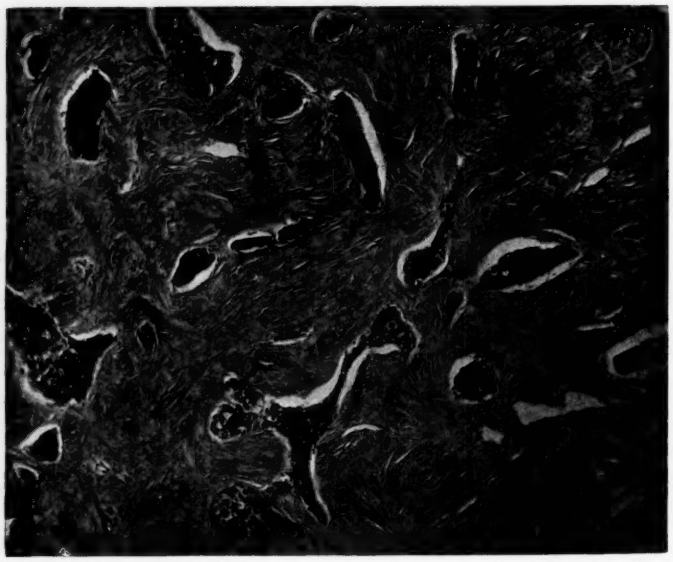

FIG. 419.—*Case* 1. Section of extreme spreading edge of tumour, showing dissemination by permeation of lymphatics. (Obj. Leitz 6a, Oc. × 10. Enlarged × 2.)

thick, uniformly surrounding the urethra from the triangular ligament to the root of the penis. The urethra is reduced to a narrow tortuous channel

lying in the centre of the growth. Sections show irregular masses of transitional-celled carcinoma with abundant fibrous interstitial tissue. At the extreme edge the tumour appears to be spreading by lymphatic permeation. (*Figs.* 418, 419.)

Case 2.—Age 59. When 21 years old, whilst in the army, the patient contracted gonorrhœa, for which he was then treated. Six years ago he commenced to have difficulty in passing urine. Because of this symptom he attended hospital, and was informed that he had a stricture of his urethra. Bougies were then passed at varying intervals during the next five years on 111 occasions; the total number of instruments actually passed along his urethra was 477. About twelve months ago the patient noticed a small hard nodule about the size of a pea on the left side of the penis at the penoscrotal junction; thereafter more and more pain was experienced with the passage of instruments. In the intervals he had marked urethral pain during micturition and a slight discharge.

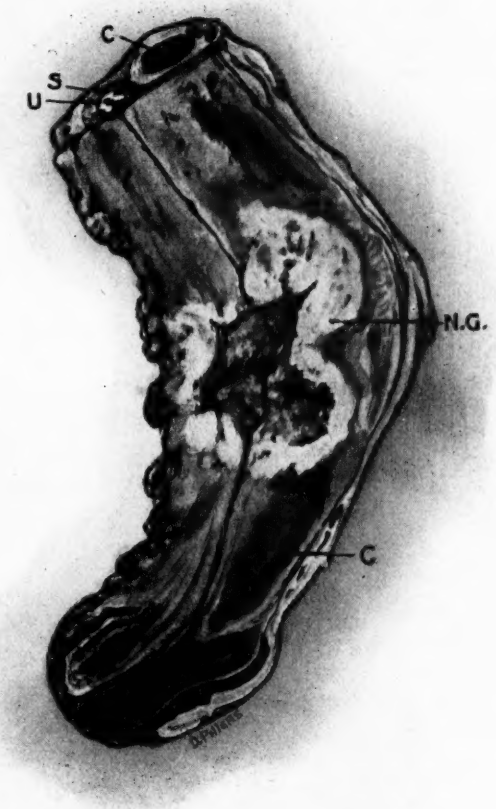

FIG. 420.—*Case* 2. Longitudinal section of penis. C, Corpus cavernosum; S, Corpus spongiosum; U, Urethra; N.G., New growth.

Seven months went by; it became impossible to pass instruments through the stricture, and the patient was admitted to hospital for operative treatment. He looked well and healthy, had a good appetite, but was losing weight.

ON EXAMINATION.—A hard stricture was palpable at the penoscrotal junction, with a nodule about the size of a small marble on its upper and left side. Malign change in the stricture was not suspected.

FIRST OPERATION.—Complete excision of the stricture was performed, and a pathological examination of the tissue removed was made. It was found to consist of scar tissue, extensively infiltrated by transitional-celled epithelioma. The patient made an excellent recovery from this operation and steadily put on weight to the extent of 2 st. 1 lb. Micturition was normal and painless. He was so much relieved from his pain and discomfort that he returned to work and refused to consider any further operation at that time.

Two and a half months later a nodule again became palpable at the site of the operation. At no time had glands been enlarged in his groins, nor could any now be felt. Immediately following this examination an abscess

developed in the region of the operation, which on bursting discharged pus and urine for about one week. The sinus then closed and micturition again became normal. Seen one month later a large mass was palpable extending

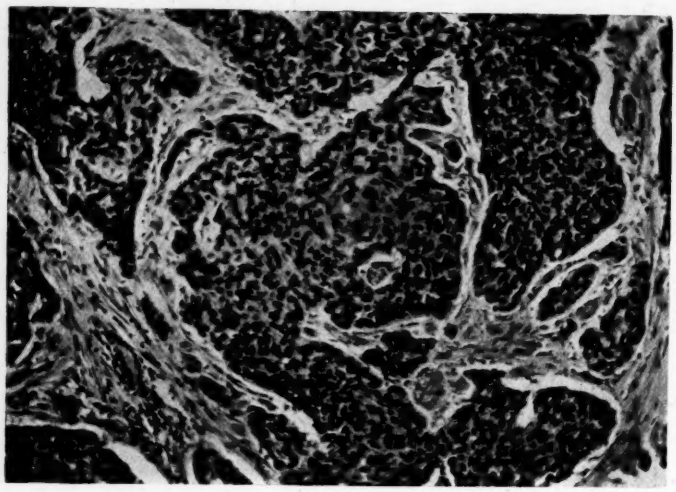

FIG. 421.—*Case* 2. Microscopic appearance.

from the membranous urethra to about half an inch distal to the penoscrotal junction. Further operation was recommended.

SECOND OPERATION.—Complete amputation of penis and scrotum (one testicle being placed in the inguinal canal) was performed. The inguinal glands still remained unaffected, and their removal was postponed to a later date.

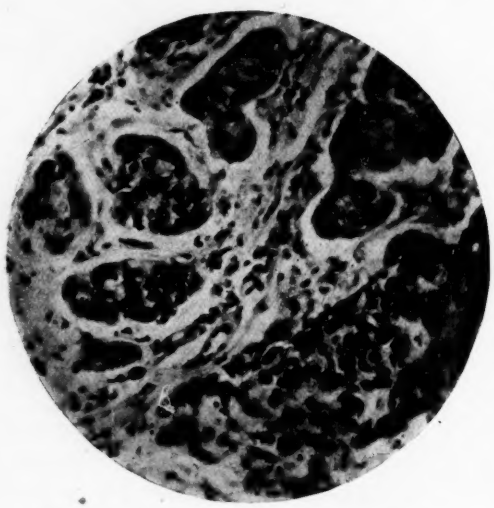

FIG. 422.—*Case* 2. Microscopic appearance.

Since this operation the patient's robust appearance has not altered and his appetite is good.

EXAMINATION OF SPECIMEN (*Fig.* 420).—The penis was cut longitudinally. It is apparent that recurrence of the growth has taken place in the situation from which the stricture was excised; the main bulk of the tumour is growing into and expanding the lumen of the urethra. Extending in an outward direction it involves the corpus spongiosum urethræ and the corpus cavernosum penis.

The sections of the growth (Dr. Hadfield) "show that it is a transitional-celled carcinoma infiltrating the tissues of the penis to within a few millimetres of the scrotal skin. It is cellular and rapidly growing: its cells are in close apposition, grouped into

large masses, and show many mitoses. A few foci of imperfect rudimentary keratinization are present. At the growing edge there is a fairly vigorous stroma reaction, and here the cell masses are smaller. Except at the edge of the central cavity there is surprisingly little necrosis for such a rapidly growing tumour. The central cavity is lined entirely by necrotic growth and shows no metaplastic phenomena." (*Figs.* 421, 422.)

Before proceeding to a short survey of the subject of malignant disease of the male urethra it may be interesting to draw attention to the outstanding features in this particular case. These are the long history of instrumental treatment of the stricture, with the inevitable constant irritation; the change from an innocent to a malignant stricture, with the difficulty and delay in its recognition; the slight effect produced in the general health and appearance of the patient; the incidence of a transitional-celled carcinoma in the corpus spongiosum urethræ. Again, it is unique in that it was proved to be a malign change in a stricture of the urethra.

No less abstruse than the etiology of most carcinomas is the etiology of carcinoma of the male urethra, but several factors may be regarded as predisposing to it. Distinctly it is a disease of advanced age, the majority of cases occurring between the ages of 50 and 60; the youngest recorded case is 38 (Kopke) and the oldest 74 (Peacock). Pre-existing urethral infection is suggested as another factor, but on this point a variety of opinion exists. Kretschmer declares that a gonococcal infection plays little or no part in the production of malignant disease of the urethra, substantiating his opinion with the observation that, though stricture of the urethra is common, malignant disease is rare. Rizzie, on the other hand, in a collection of 35 cases, states that 50 per cent gave a history indicating the presence of stricture of the urethra.

At this stage it may fairly be noted that mere history unsupported by actual proof of the presence of stricture is a doubtful quantity upon which to base opinion, as the earliest symptom of carcinoma of the male urethra is also that of stricture. The second case presented in this article is the only one we can find recorded where stricture with a superadded carcinoma has been definitely proved. It may therefore be deduced that a gonococcal urethritis is not an immediate factor in the etiology of malign neoplasm in the male urethra.

As in other parts of the body, papillomata of the urethra may undergo malignant changes. The part taken by chronic irritation in the production of cancer may be evidenced in the present case—477 passages by instruments were made through the stricture. As a further example of irritation the case recorded by Kretschmer may be quoted, where he maintains that an injection of Hartzell's solution into the urethra forced a carcinoma into activity. Lastly, we again meet that interesting phenomenon, the predisposition of a developmentally displaced epithelium to awaken into unrestricted growth. Rare though transitional-celled carcinoma of the penile urethra appears to be, embryologically it would seem to be a simple transposition of cells. According to the work of Kiebel and Mall, the bladder and urethra as far as

the corona glandis are cloacogenic and mesodermal in origin. The tendency of cytological change towards malignancy in the mucous membrane of the urethra as compared with its histology forms an interesting observation. Transitional-celled epithelium lines the prostatic urethra, columnar-celled the membranous and penile urethra, and only the fossa navicularis is squamous.

From 76 cases collected from literature in which evidence as to the nature of the tumour is available, the following list is compiled: squamous-celled carcinoma, 73 per cent; columnar-celled carcinoma, 1·5 per cent; papillary carcinoma, 3·5 per cent; adenocarcinoma, 21·2 per cent (from Cowper's glands); transitional-celled carcinoma, 1·3 per cent.

As regards localization of these tumours, great difficulty is experienced owing to insufficient data in recorded cases. Reference therefore will merely be made to the 61 cases collected by Diehle and classified thus: membranous urethra, 33; pars cavernosa, 26; fossa navicularis, 2. Recognizing the high percentage of squamous-celled carcinomata which occurs in the male urethra makes one cast a doubt on the small number which take origin in the fossa navicularis. Descriptions are inadequate to make this opinion decisive, but quite a large proportion of those involving the anterior part of the pars cavernosa may take origin in and around the fossa navicularis and thence spread backwards.

It is only in very rare instances that cancer of the urethra is diagnosed in its early stages; the majority of cases pass through the stage of treatment as strictures of the urethra, and not till late is the proper label affixed and adequate treatment begun. It would be true wisdom were every pathological change towards tumour formation in the urethra immediately suspected as tending towards malignancy and treated as such.

The symptoms of the disease may be divided into three groups or stages. In the first stage, as in stricture, there is difficulty with micturition from the early obstruction by the tumour, a difficulty which is usually accompanied by urethral pain. Most often a discharge is present, purulent or, it may be, blood-stained. Retention of urine sooner or later occurs, and the picture immediately suggests simple stricture. The patient looks well, but as a rule feels weak. This stage may extend into several months. In the second stage a local tumour forms. In the third stage infection is the predominating factor; peri-urethral abscess forms, which on bursting results in fistula formation. In 80 per cent of cases this fistula opens on the under surface of the penis.

For the purpose of estimating prognosis, 36 cases have been collected. Of these, 25 per cent were dead in the first year, 19·4 per cent had recurred in the first six months, and 55·6 per cent had not recurred at the end of the first year. Diehl in his article makes an interesting remark on prognosis: "Krebs der Pars pendula Urethræ sind prognostisch günstigst aufzufassen als solche der Pars fixa."

No case is recorded where the tumour recurred in the inguinal glands,* these only becoming affected when the growth has involved the scrotum and surrounding skin. The operation of choice, however, has been that described

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* *Vide* terminal note

by Pearce Gould (*Lancet*, 1882, i, 821), with removal of the inguinal glands. One case—namely, that of Culver and Forster, of Chicago—may be quoted where the inguinal glands were not removed, and six months later there was no sign of recurrence and the patient was in good heath.

I wish to express my indebtedness to Professor E. W. Hey Groves, with whose kind permission I have recorded the details of these cases.

NOTE.—The patient in *Case* 2 was readmitted to the hospital in January, 1928, and the inguinal glands were dissected out from both sides. These showed abundant evidence of malignant disease.

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PATELLA CUBITI.

By GEORGE GUNN,

ASSISTANT PHYSICIAN TO THE ROYAL LIVERPOOL CHILDREN'S INFIRMARY.

In December, 1926, I was asked to see S. R., age 46, a woman who had been knocked down by a bicycle the previous evening. On examination there was apparent a severe injury to the left elbow-joint, with marked pain, swelling, and immobility. A loose fragment of bone was felt above the position of the olecranon. The injury was diagnosed as fracture of the olecranon with separation of the fragments.

Fig. 423.—Skiagram taken four days after injury.

Twenty-four hours later the swelling was somewhat reduced ; the movable fragment was not painful to handle, no sharp edge could be felt, both ends of the fragment were smooth and rounded, no crepitus could be elicited, and the continuity of the triceps tendon was not interrupted. The joint was still very swollen and tender, but the point of maximum tenderness was about one inch below the upper end of the ulna. A skiagram taken four days after the injury (*Fig.* 423) showed a fracture of the olecranon, and the abnormality now to be described.

Four months after the injury (*Fig.* 424) the patient had made an excellent recovery. Full flexion is now obtainable with ease, extension is present to 160°, and there is no interference with pronation and supination. In the olecranon fossa a bony mass is present, $\frac{3}{4}$ in. in width and $1\frac{1}{4}$ in. in length. Lying within the fossa this bony mass is only slightly prominent above the contour of the limb. The 'feel' of this body resembles that of a patella, a similarity which is accentuated by the free lateral movement possible on relaxation of the triceps tendon. The shape is roughly ovoid, and at the widest part is marked by a definite transverse ridge. This ridge is at the junction of the lower and middle thirds.

In 1903 Kienboch[1] showed a man who had a large patella-like sesamoid bone close above either olecranon. This man had sustained an injury to the left elbow in his seventeenth year, and a later injury (year unstated) to the right elbow. Katz[2] in 1915 referred to Kienboch's paper, and adds the case of a 37-year-old soldier who had a plexus paralysis of the right arm. " On

the extensor surface of the right arm, buried in the triceps muscle, lay a piece of bone stretching from the point of the olecranon to the distal end of the humerus and measuring 4 cm. in length by 1 cm. in breadth. The bone is sickle-shaped and stretches from one articular surface to another. The bone abnormality is one sided only." Katz proceeds to quote Virchow and Pfitzner as accepting these cases as sesamoid bone, but personally follows Kienboch's views that they are of traumatic origin. Japiot[3] says: "La bilatéralité de noyau osseux—accolé à la paroi de la fosse olécranienne—il s'agit donc d'un sésamoïde olécranien, c'est-à-dire d'une abnormalité d'une extrême rareté." Cunningham[4] remarks that in one recorded case the olecranon was a separate bone.

The idea of the patella possessing a homologue in the elbow region was first mentioned in European anatomy, according to Eisler[5], by Vicq d'Azyr in 1774, and by Meckel in 1916. But Hoernle[6] shows that the Hindu anatomists four or five hundred years B.C. were imbued with a similar idea, and he writes: "*Kapalika* or *kurpara* (elbow pan), and *janu* or *januka* (knee cap), are the olecranon process of the elbow, and the patella of the knee. The former is not a separate bone. But by the ancient Indian anatomists it is counted as a separate bone. The term *kurpara* is peculiar to Susrata, who expressly defines it as denoting the homologue of *janu*, the knee cap. The term *kapalika* is peculiar to Atreya; it means literally a small dish, and is therefore identical in meaning with *patella*, the Latin name for knee cap."

FIG. 424.—Skiagram of left elbow, lateral view. Four months after injury.

Reeves[7] quotes Gegenbaur as advancing the view that the olecranon corresponds to the patella, but Quain[8] holds that the patella has no homologue in the human body. Patterson[9] quotes a case from Agayeff: absence of the inner half of the forearm and hand. The humerus was normal, and in the trochlear region was a round bony prominence of the shape of the patella, movable in all directions and attached to the triceps muscle. The Chiroptera are described by Flower[10] as the only mammals which constantly have a detached separate ossicle in the triceps tendon. Parsons[11] holds that the epiphysis of the upper end of the olecranon is an exception to the generalization that traction epiphyses are peculiar to mammals. He instances a Surinam water toad which shows a sesamoid in the triceps tendon just above the ulnar insertion. This sesamoid is frequently found in the Reptilia, but in closer relation to the ulna. In mammals he further states that this sesamoid is represented by the epiphysis for the upper end of the ulna—except in the bats, when it remains separate, and is attached to the ulna by fibrous tissue; it therefore closely resembles the patella of other mammals. In man the epiphysis has

usually the mammalian arrangement, but it may form quite half the olecranon and encroach on the articular surfaces, although cases are recorded in which it fails to unite with the olecranon, as in bats.

The ossification of the upper end of the ulna is described by Quain[12] as being from a centre appearing about the tenth year, and uniting at the seventeenth. Gegenbaur,[13] Gray,[14] and Morris[15] agree with this. Fawcett[16] describes two ossific centres for the olecranon, and Cunningham[17] says that an independent centre of ossification for the extreme edge of the olecranon has been described.

Professor Robinson, of Edinburgh University, very kindly read my notes on this case, and informs me that the single centre for the olecranon is most common; that this single centre may form about one-half the olecranon, the remainder being ossified from the shaft, or it may form a shale of bone on the upper and back part of the olecranon which takes no part in the formation of the articular surfaces. The centre for this shale may be accompanied by an additional centre of ossification placed above and in front of it; this additional centre forms the apex of the olecranon and the adjacent part of the articular surface. The single centre is the most common: it forms the shale mentioned in 50 per cent of cases.

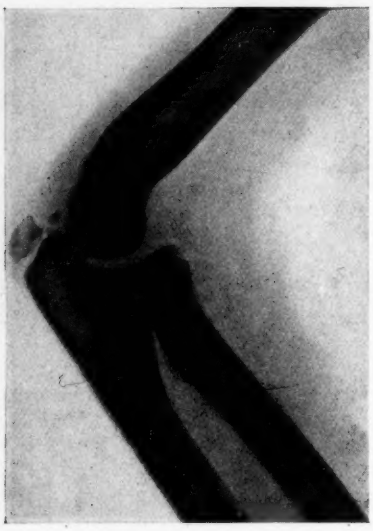

FIG. 425.—Dr. MacFarland's patient.

Professor Robinson further states that the olecranon can scarcely be homologized with the patella, because the ossific centre appears in a part of the cartilage of the ulna, not in a new-formed piece of cartilage in a tendon; and the fact that the epiphysis remains separate does not alter the essential fact that the centre of the ossification was found in the cartilage of the ulna. Apparently this case is one of persistent separation of the olecranon epiphysis. This view is supported by measurements of the photographs. The distance from the tip of the coronoid to the tip of the olecranon is greater on the injured than on the normal side. This may be accounted for by fracture, but Professor Robinson states that the distance is greater than in a series of normal bones; and he also points out that the height of the olecranon as measured from the proximal surface of the coronoid to the apex of the olecranon is greater on the injured side, and also greater than the height of a series of normal olecranons.

Sir Robert Jones, whom I also consulted, tells me that in his vast experience he has not seen a similar case. Sir Robert expressed the opinion that the condition was probably due to an earlier injury; but the history is negative on this point, and in view of the anatomical opinion expressed by Professor Robinson I think that the diagnosis of persistent separation of the olecranon epiphysis must be adhered to. In support of this view the X-ray photograph

lent me by Dr. MacFarland of a boy 12 years of age showing three separate ossific centres is of interest (*Fig.* 425).

I am very much indebted to Mr. Bryan MacFarland, of Liverpool, and Professor Robinson, of Edinburgh, for kindly help in my researches.

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KÜMMELL'S DISEASE.

By J. CARDIS, G. F. WALKER, and R. H. OLVER,

THE GENERAL INFIRMARY, LEEDS.

In a paper read in Hamburg in 1894 Kümmell described a disease which he said had been 'hitherto unrecognized'. He had formerly reported four cases to a meeting at Halle in 1891. He wrote descriptive papers in 1895 and again in 1921. In 1911 his pupil Schultz collected and described an important series of twenty-one cases. Kümmell's disease is still struggling for recognition, and the object of this paper is to describe the disease with illustrative cases. It may be defined as the delayed crumpling collapse of the body of a vertebra following injury.

Schede in 1881 drew attention to a disease characterized by the slow softening of the body of a vertebra followed by the late development of an angular kyphosis. König also mentioned the disease in a contemporary text-book, but he regarded it as an atypical form of spinal tuberculosis. At first Kümmell thought that the disease was independent of fracture, but he altered his opinion before his 1895 paper. In 1892 Verneuil described the malady, and in 1897 Schneller reported a case of the disease following indirect injury. Reuter wrote on the pathology of the disease in 1894, and a later reference will be made to his work. The important case series of Schultz and Kümmell himself have already been mentioned, but the biggest article on the subject available at present for ready reference by English-speaking surgeons is the paper by Fosdick Jones in 1923, entitled, "Compression Fracture of the Spine: Post-traumatic Spondylitis, or Kümmell's Disease". In this paper, Jones contributed an important case to the literature and gave an exhaustive review of other published cases, with a full bibliography.

Type Case.—Following an injury, direct or indirect, to the spine, a man may be incapacitated for a little while owing to bruising, with diffuse or localized pain, and in the more severe cases girdle and limb pains. There may also be locomotor trouble ranging from slight leg weakness to total paraplegia with sphincter paralysis. After a short interval these symptoms regress, and soon the man is apparently well enough to resume his ordinary occupation. After a period lasting for weeks or even years the man experiences a return of the pain, and sometimes a return of the symptoms referable to cord interference. On examination he is found to have a spinal deformity, usually, though not invariably, angular in type. This syndrome advances, but is amenable to treatment and can be arrested.

Course of the Disease.—

1. *The Injury* varies greatly in type and severity. In the first place there may be direct injury to the back, as in the case of a miner buried by a fall of 'roof', or a man falling from a scaffolding and 'landing' on his back. On the other hand, there may be a mere jarring of the spine caused by a jump from a

moving vehicle. In the one case there may be a fracture with or without dislocation, whilst in the other there may be no demonstrable injury at all. Balthazard pointed out that, in cases where a fracture is from the first demonstrable, it is usually in the anterior part of the body of the vertebra. Cord interference therefore in the first stage is due to concussion.

2. *The Post-traumatic Interval* likewise varies greatly. The man may be confined to bed on account of contusions, he may be paraplegic, or he may feel no ill-effects whatever. There is usually, though not invariably, some pain at the site of the vertebra selected for ultimate destruction.

3. *The Latent Period* between the cessation of the original symptoms and the onset of severe and progressive disability is noteworthy for several reasons. It may be extremely short, amounting to an amelioration rather than a cessation proper. It may be days, weeks, months, or years, during which time the subject is conscious of no malady whatever. Indeed, the future cripple may resume heavy work. The medico-legal aspects of this latent period will not be ignored by those whose duties bring them into contact with the Workmen's Compensation Act.

4. *The Return of the Symptoms* leads the man to focus his attention on his former spinal injury. The returning symptoms at first are local—namely, pain, tenderness, and weakness at the site of the lesion ; but remote symptoms —namely, all grades of limb and girdle pain and all grades of paraplegia—tend to supervene.

5. *The Fifth Stage* presents one of two possibilities. Either the crumpling collapse of the vertebral body is arrested by orthopædic measures, in which case the prognosis is singularly good, or the process goes on till the cord is transversely crushed.

Basic Pathology.—Two hypotheses have been propounded. The first is that the crumpling collapse of the vertebral body, during which it assumes a wedge shape, follows a lack of nutrition consequent on local injury to small vessels with focal hæmorrhages and thromboses. The second is that the change is a trophic disturbance set up by injury to the nerves and the local part of the cord. Originally Kümmell considered that the process was a 'rarefying osteitis' of inflammatory origin following nutritional disorder. He later amended his views in so far as he came to the conclusion that damage to the bone was always inflicted by the original injury, though not always demonstrable. In 1896 Mikulicz put forward the view that the original trauma caused intradural and extradural hæmatomata, and that these were accompanied by infiltrations of the nerve-roots and ganglia, whence trophic bone mischief arose. Henle contended that the trouble was of vasomotor origin and that the case showing vertebral fracture was exceptional. In 1904 Reuter reported " luxation of the bony trabeculæ and extensive hæmorrhage into the bony cellular tissue ". Ludloff at the Dresden Orthopædic Congress in 1920 reported that, in a case in which there had been trauma but no demonstrable fracture, he had found rupture of the vessels of the lumbar segments associated with contusion of the bodies and nutritional impairment. All this led to necrosis of the anterior parts of several lumbar bodies and consequent kyphosis. In 1921 Kümmell spoke of " atrophy of the bony framework and spongiosa following trauma ". Arising out of the discussion after Fosdick Jones's paper

in 1923 came the general opinion as to the essential pathology—" that minute multiple traumata to the osseous and ligamentous structures of the spine caused cracks and small hæmorrhages ".

As illustrating the generally accepted pathology we quote the following case :—

Case 1.—J. H., age 24. In *September*, 1924, he had a fall of stone on his back ; was detained several weeks in hospital, but no spinal injury was detected by radiography or otherwise. In *April*, 1925, he complained of pain in the lower part of the back, and marked deformity of the lumbar spine was discovered Radiography showed narrowing of the discs between the 1st and 2nd and between the 2nd and 3rd lumbar vertebræ. There was some absorption of the body of the 2nd lumbar, and it had assumed a wedge shape. A provisional diagnosis of spinal caries was made, but by chance the man's death from an intercurrent malady later in the year made possible the following examination of the affected vertebral bodies.

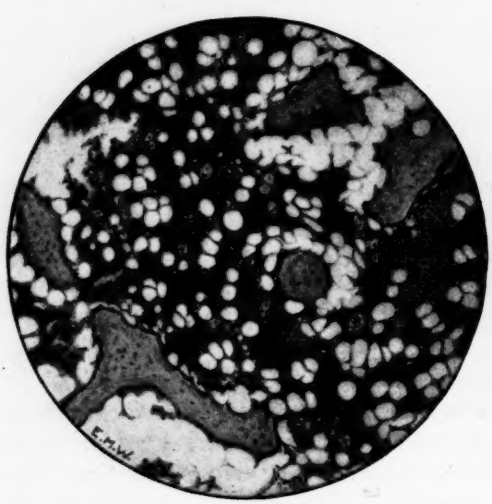

FIG. 426.—Microscopic appearance of normal vertebral body structure.

FIG. 427.—Microscopic appearance of Kümmell's disease.

" No signs of spinal cord injury. No evidence of tuberculosis or abscess. No appearance of necrosis, but the 2nd lumbar body was wedge-shaped. The intervertebral discs were healthy, and the margins of the bones were intact except for a small fracture in the upper anterior part of the 2nd lumbar body. On sagittal section none of the vertebræ appeared to have lost much in the way of density, though the 2nd was wedge-shaped and the spongiosa showed multiple small hæmorrhages. Microscopical examination of a sagittal section of the middle third of the 2nd lumbar body showed marked atrophy of the bony framework, with a changed appearance of the marrow, the cells of which were decreased and stained poorly. *Fig.* 427 shows this atrophy and diminished cellularity."

In view of these findings, and in spite of the scanty history, we regard this case as one of Kümmell's disease, illustrating pathologically : (1) Slight fracture at the time of injury ; (2) Small focal hæmorrhages in the spongiosa ; (3) Atrophy of the bony framework and deterioration of the bone-marrow ; (4) Assumption of a wedge shape by the affected vertebral body (*Fig.* 428).

It has been shown by Lange that, in a vertebral body experimentally injured so as to produce nutritional impairment, the crushing by the weight of the spine above tends to cause a wedge shape.

Diagnosis.—Both from a medico-legal and prognostic aspect early diagnosis is of paramount importance, but the difficulties are great, especially in the case of slight injury. The diagnosis of Kümmell's disease should always obtrude in cases of slight injury in which there is undue persistence or recurrence of symptoms. Radiography is all-important, though a negative finding is inconclusive in the early stages. A lateral view is indispensable. In differential diagnosis the following lesions have to be considered: simple contusions, strains, neuroses, traumatic myelitis, syphilitic spondylitis, rheumatoid arthritis, and Pott's disease. The differentiation between Pott's disease and Kümmell's disease presents real difficulty, but we would emphasize the facts that in the latter: (1) The sufferer is in all other respects a healthy robust workman; (2) There is no abscess formation; (3) Radiology shows a wedge-shaped vertebra, with possibly a fracture, but with clear edges and intact discs.

FIG. 428.—*Case* 1. Drawing of a sagittal section through the lumbar spine, showing the wedge shape of the 2nd lumbar vertebra.

Treatment and Prognosis.—Only the broadest principles can be laid down. It is essential to secure rest to the spine to relieve the pressure on the crushed body. Extension of the spine, with submission perhaps to a Bradford frame, may be called for in some cases. Support to the spine by some form of corset or plaster jacket worn continuously for long periods is useful. Spinal bone-graft has been employed with advantage. Ankylosis of the spine and welding by callus are aimed at, though the former is rarely attained.

The ultimate prognosis as to cessation of local and remote pain and relief from cord interference, together with restoration of back strength and general rehabilitation of the patient, would seem to be very good. We have noticed the installation of true neurasthenia in some patients and apathy as to progress. The difficulty in getting a workman to give up a supporting jacket once he has taken to it is often very great. If a man is back at work within two years from the original injury, he is very fortunate, and so are his advisers.

CASE RECORDS.

Case 2.—W. B., miner, age 27. In *December*, 1923, he was struck on the back by a fall of 'roof' and was taken home to bed suffering from pain in the back and shock. The pain in the back and bruising were gone in a day or two, but he was kept in bed as a precaution, and while resting in bed he noticed a 'lump' in the lower spine. He sought hospital advice in *March*, 1924, on account of the lump. He had no symptoms. A diagnosis of probable spinal caries was made, and a bone-graft operation was done forthwith. One of us saw him again in *September*, 1926. He was working as an engine-driver, and was remarkably free from disability or

pain. An X-ray plate taken at this time (*Fig.* 429) shows a clear wedge-shape appearance of the 1st lumbar vertebra. There is no sign of tuberculosis. In view of the history and the subsequent good health, we dissent from the original diagnosis of spinal caries and present this as a case of Kümmell's disease.

FIG. 429.—*Case* 2. Radiogram taken three years after original injury.

FIG. 430.—*Case* 3. Contour of lower dorsal spine in December, 1924.

Case 3.—A. C., age 40. Was seen early in 1924 for furunculosis and had his back examined, but no kyphosis was detected. *March*: Whilst he was in a kneeling position a fall of stone pinned him and crushed him in an antero-posterior direction. He was kept in bed for nine weeks on account of lower dorsal pain. *May*: Advised to do light work and certified independently as fit to do so. Began complaining persistently about lower dorsal pain again. *October*: Again independently advised to do light work and had his compensation stopped. Tried to work and could not, on account of pain in the back. *December*: Case re-opened. 11th dorsal vertebra found to be collapsed and to have assumed a wedge shape. Given a Thomas support for his back. (*Fig.* 430.) *July*, 1926: Seen by one of us. Found to be free from pain in the back and able to do light work. The Thomas support appeared to have arrested the collapse of the 11th body and to have brought about an amelioration of all symptoms with a general rehabilitation.

Case 4.—T. B., age 29, mine worker. *May*, 1924: Fell 20 feet from a ladder and struck his back on a rail. Was concussed and kept in hospital twelve days. Walked home safely and could walk well and was free from serious disability for several months. *Midsummer*: Started to complain of pain in the back of increasing severity, also of early weakness of legs. *September*: Began to be disabled by pain in the back and weakness of the legs. Found to have a tender lump on the back and a marked angular kyphosis involving D11, D12, L1, and L2. There was definite motor weakness of the legs. Radiology showed absorption of the 1st lumbar body, with the assumption of a wedge shape. A diagnosis of Kümmell's disease was made and a supporting jacket supplied. Steady relief of the dorsal pain was experienced, and the leg weakness disappeared in the course of several months. *September*, 1926: Seen by one of us. Spine firmly stationary and healed. No progress of the collapse noted in 1924. Able to get about well. Fit for light work. Free from dorsal pain and leg weakness. *September*, 1927: Seen by another of us. Found to be well and strong, and still wearing the jacket, though obviously unnecessarily. Malingering.

Case 5.—J. F., age 26. *November*, 1923: Struck heavily on the back by a stone. Brought to hospital severely injured. Placed on a Bradford frame, on which he lay for nine weeks, during which time he experienced spasmodic contractions of the leg muscles and a throbbing pain in the back. *January*, 1924: At home, better. Merely felt that his back was 'not quite right'. Noticed a 'lump' on his back for the first time. *May*, 1924: Kümmell's disease diagnosed and spinal bone-graft done. *July*, 1926: Reviewed by one of us. Found to be in sound health. Still wore a spinal support, and had no back pain or leg weakness connected with the accident. *Fig.* 431 shows the permanent contour of the 3rd lumbar body in 1926.

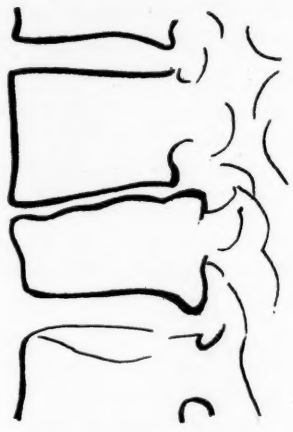

FIG. 431.—*Case* 5.

FIG. 432.—*Case* 6.

Case 6.—J. C., age 39. Was severly injured by a fall of stone to his back while working in the mine. Found to have a fracture-dislocation of the 1st lumbar body with slight wedge-shape assumption (*Fig.* 432). Local pain and local cord interference are subsiding under treatment by his immobilization on a Bradford frame.

Case 7.—J. O., age 54. Early in 1925 was struck on the back by a falling stone. Kept in bed for several weeks on account of pain and shock. *Mid*-1925: Much relieved. Up and about. Still having slight pain at the site of injury. *Late* 1925: Exacerbation of dorsal pain. Found to have multiple healed scars on the back and a kyphosis involving the 12th dorsal vertebra. Much local tenderness. Much pain and limitation of movements of the spine on attempting to extend the spine. X rays showed well-marked absorption of the 1st lumbar body. Fitted with a spinal jacket and relieved. Insanity from another cause supervened in this case, but we hear that his spinal condition is stationary without symptoms.

Case 8.—G. N. F., age 28. While riding a motor cycle was thrust several feet rightwards by a collision. The circumstances of the collision call for special comment. The patient was struck on the left foot by a car which unexpectedly emerged from a side street on his left. Only the patient's foot was struck. His back was not directly injured, and he was not unseated. He was certainly

FIG. 433.—*Case* 8. The contour of the lumbar spine taken a few weeks after the patient's *indirect* injury.

jarred severely, but he resumed a twelve-mile journey on his bicycle. One week after the accident the patient noticed a lump in the 'small of the back' and associated pain and tenderness. A skiagram showed wedge-shaped collapse of the 2nd lumbar body (*Fig.* 433). The pain and tenderness were relieved by a supporting jacket, and the patient is now not functionally disabled in any way and has no symptoms. We regard this as an extremely important case.

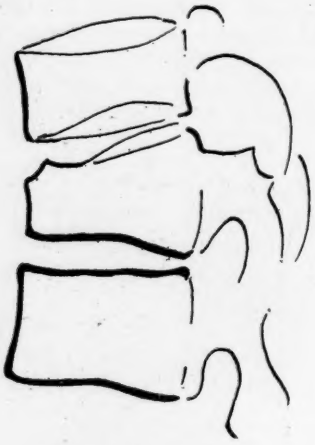

FIG. 434.—*Case* 9.

Case 9.—J. C. K. Was struck on the shoulder by a heavy iron casting. His spine was acutely flexed at once, and there was intense immediate pain in the lower dorsal region. The X-ray plate, of which *Fig.* 434 shows the contour, revealed a slight fissured fracture in the 12th dorsal body, but we note the wedge-shape appearance of the body in addition. We have not been able to trace this patient, but when one of us last saw him he was under Bradford-frame régime and feeling comfortable.

Case 10.—H. H. Was an occupant of a falling cage in the pit. When the cage fell to the bottom of the shaft, several who were in it were killed and the patient was jarred from heel to crown. When he arrived in hospital he was still concussed and did not recover for some days. He passed immediately into a condition of paraplegia and cystitis, and died two months after admission.

The post-mortem examination revealed crushing and atrophy of the 2nd lumbar vertebra with multiple small fractures in it. *Fig.* 435 is a drawing of the pathological specimen.

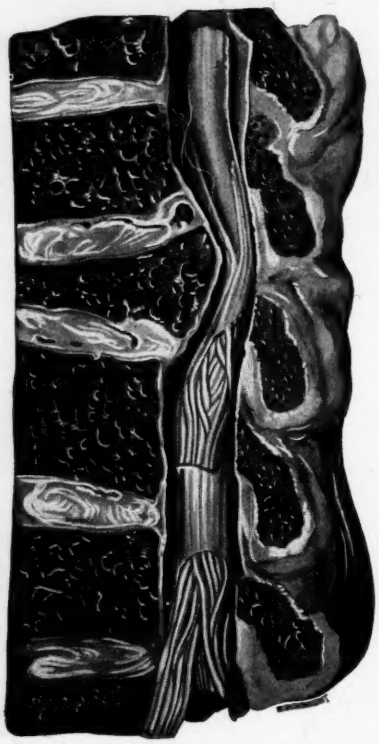

FIG. 435.—*Case* 10. Pathological specimen showing the collapse of the vertebral body and the cord interference.

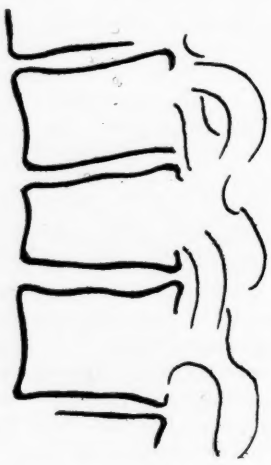

FIG. 436.—*Case* 11.

Case 11.—J. M. Was struck on the back by a fall of 'roof' in the mine. Was in bed for several months owing to shock and bruises. Enjoyed fair health with no real disability for a subsequent period of about four months. Next he became gradually incapacitated by pain in the back, girdle pains in the trunk, and finally shooting pains down the legs. *Fig.* 436 shows the contour of the 12th dorsal vertebra at the time when he came for treatment. A poroplastic jacket relieved the local pain and remote pains, and restored to him his ability to work.

FIG. 437.—*Case* 12. The cervical spine, showing the assumption of a wedge shape by two adjacent vertebræ as a result of an accident twenty-five years before death.

FIG. 438.—*Case* 13.

Case 12.—T. H. D., age 40. We think this is a unique case in the literature. The patient, when a boy, experienced an injury which was not fully revealed until adult life. The story is as follows: At the age of 15 he was struck on the back of the neck by a runaway truck. His injuries were severe and he was paraplegic for no fewer than nine years. He recovered, however, and took part in the great war. At the age of 40 death occurred from cystitis associated with paraplegia due to a wholly independent lower dorsal spinal cord tumour.

Fig. 437 is a drawing of a sagittal section through the cervical spine at the time of the post-mortem. The lesion corresponds with the site described by the patient as the site of his injury in youth. On dissecting down to the lesion we found a certain definite callus formation in the spines, confirming the story of the injury, and we present this case as one of Kümmell's disease affecting two adjacent cervical vertebræ. Macroscopically the bodies were sharply and cleanly wedge-shaped.

Case 13.—A. W. Fell several feet and 'landed' heavily on his shoulders. Went to bed for several weeks on account of extensive backache. His pain persisted in the lower dorsal region after he was able to get up, and it gradually became worse

and made him take to his bed again. A radiograph shows a fracture of D12 and an associated slight crumpling of L1 (*Fig.* 438.) This patient is still on a Bradford frame, and while on it he is free from pain.

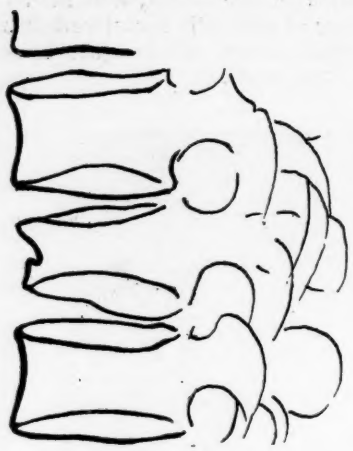

FIG. 439.—*Case* 14.

Case 14.—R. S. Was struck on the shoulders and experienced acute flexion of the lumbar spine. A skiagram taken shortly afterwards shows collapse of the 1st lumbar body. (*Fig.* 439.) This patient is under treatment on the usual lines.

SUMMARY OF KÜMMELL'S ORIGINAL CASES.

INJURY	LATENT PERIOD	SITE	RESULT
Jump to a platform from a a moving train	1 year	D7 and D8	Recovery
Thrown forward on his knees by a sack of flour ..	7 months	D6	Resumption of heavy work
Fall from a ladder.. ..	5 months	D6	Ditto
Fall backwards on some stairs	18 months	D3 and D4	Ditto
Fall from a horse	9 months	D6 and D7	Resumption of duties as a cavalryman
Fall from a low window ..	10 weeks	D6 and D7	No permanent ill-effects

As this paper is concerned with a syndrome hitherto outside the ordinary text-books, we think we may usefully raise a few questions rather than comment on our cases.

1. Does Kümmell's disease ever affect two vertebræ which are not contiguous ?

2. Is the disease more common than has been supposed ?

3. What is the real determining factor bringing about the selection and destruction of a vertebral body ? (*See Case* 8.)

4. What is the general experience as to prognosis ?

5. What analogous changes in other bones can be adduced to clear up the essential pathology of Kümmell's disease ?

We acknowledge with thanks our indebtedness to the honorary staff of the General Infirmary at Leeds for permission thus to record their cases. In particular we thank Dr. Telling, Dr. Watson, Mr. Daw, Mr. Collinson, and Professor Stewart. It was Dr. Telling, to whom each of us has been attached as house physician, who suggested this paper. We have been materially assisted by Mr. L. R. Braithwaite, Director of Surgical Research in the University of Leeds.

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CHRONIC PEPTIC ULCER OF THE STOMACH WITH ACUTE MILIARY TUBERCULOSIS OF THE GASTRIC MUCOSA.

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Tuberculous infection of the stomach is rare. In the Mayo Clinic it was encountered only once in a consecutive series of 2501 gastric operations performed during the years 1912 to 1915 inclusive (Broders,[1] 1917). Melchior[2] (1926), who has recently reviewed the literature of *surgical* tuberculosis of the stomach, reports 6 cases observed in Kuttner's clinic during a period of ten years, and expresses the opinion that gastric tuberculosis is more of a rarity than was formerly supposed. Broders has analysed and critically examined 305 published cases, and added one of his own. Of these, only 49 showed the presence of tubercle bacilli in the depths of the lesion, while 118 others gave a typical histological picture, but without demonstration of the causative organism. Fifty-nine cases were classified as doubtful, having either no histological details or only an inadequate report, while 80 cases were rejected altogether. Taking the 167 fully authenticated and probable cases together, Broders found an ulcerative type of lesion in 136, miliary tubercles alone in 15, and solitary tubercle or tumour in 12; 9 of the ulcerated cases showed miliary tubercles in addition.

In a consecutive series of 10,000 autopsies at the Leeds Infirmary, tuberculous ulceration of the stomach was present in three cases only, and in two of these it was due to involvement from without by adjacent tuberculous lymph glands. In a series of 320 partial gastrectomies in which the specimens were submitted to microscopic examination, none was the seat of tuberculous ulceration; but in two which are here recorded, a simple chronic ulcer was associated with scattered miliary tubercles throughout the mucosa, while in a third, also now described, typical 'carcinoma ex ulcere' coexisted with a lesion at least strongly suggestive of tubercle. The association, obviously a rare one, was present in 'a fair number of cases' in Broders' collected series, including 8 reported by two observers.

REPORTS OF THE CASES.

Case 1.—

Clinical History.—The patient, a married woman of 57, had suffered with indigestion for six years. The pain came on about an hour after food and varied in its duration. There was no vomiting in the early stages and there were considerable intervals of relief. Three years ago she had a fairly severe hæmatemesis. There had been some loss of weight, but no cough

and no sputum, nor was there any history to suggest pulmonary tuberculosis. On examination the patient was thin, and an ill-defined tumour could be palpated beneath the left costal margin. There was slight visible peristalsis. There were no definite signs in the chest. X-ray examination by Dr. L. A. Rowden revealed a large ulcer crater situated in the middle third of the lesser curvature, with slight hour-glass contraction (*Fig.* 440).

FIG. 440.—*Case* 1. The middle third of the stomach is firmly contracted down. A very large ulcer crater is seen just below the middle of the lesser curvature, its lower half filled with opaque material, its upper with air. (*Dr. L. A. Rowden.*)

At operation on April 29, 1927, a large chronic ulcer of the lesser curvature was found. It was firmly adherent to and perforating into the pancreas, which, indeed, formed a large part of its floor posteriorly. Partial gastrectomy was performed, with antecolic anastomosis between the jejunum and the cut end of the stomach. The patient made an uneventful recovery, and is now (January, 1928) in better health than she has been for years.

NAKED-EYE EXAMINATION OF SPECIMEN.—About half the stomach has been removed. There is a large chronic ulcer (*Fig.* 441) on the lesser curvature, with thickened, indurated, and undermined margins, especially towards the pylorus. The floor is smooth and hard, with a little adherent slough. There is a breach in the posterior part of the floor where the ulcer has been separated from the pancreas. Near the centre the closed stump of an artery is seen. Anteriorly, the peritoneal aspect of the ulcer (*Fig.* 442) shows much inflammatory œdema, much hæmorrhagic flecking and congestion, and a tendency for the fat of the gastro-hepatic omentum to extend downwards on the stomach wall. Posteriorly there is much fibrosis, especially around the site of attachment to the pancreas. A section through the ulcer (*Fig.* 443) shows the width of the breach in the muscular coat, the amount of undermining of the margins, and the extent and density of the fibrosis in floor and margins.

FIG. 441.—*Case* 1. The chronic peptic ulcer as seen when the excised portion of stomach had been laid open along the greater curvature. The upper line of incision is shown on the right.

HISTOLOGICAL EXAMINATION.—

The Ulcer.—Microscopically the lesion has the usual characters of a simple chronic gastric ulcer. The four

zones in the floor described by Askanazy are clearly defined: The inner purulent layer, patchily distributed, is broadest beneath the overhanging margins where it is protected against removal. The so-called fibrinoid zone, of fairly uniform thickness over the whole floor, is, as usual, deeply stained, the fibrinoid material with the acid dye, the numerous pyknotic nuclei with the basic. The third (granulation tissue) and fourth (densely fibrous) zones merge into one another and vary from $\frac{1}{8}$ in. to $\frac{1}{2}$ in. in thickness. Beyond this the fibrosis extends widely into the adjacent adipose tissue. Neutrophil leucocytes are present in abundance just below the fibrinoid zone, while eosinophil cells, plasma cells, and lymphocytes are all fairly numerous. For an ulcer of such obvious chronicity there is comparatively little change in the blood-vessels.

FIG. 442.—*Case* 1. Serosal aspect of the lesser curvature region, anterior wall, showing extension of adipose tissue over the wall of the stomach, with intense vascular engorgement and much hæmorrhagic stippling.

The Remainder of the Stomach Wall.—The chief point of interest here is the presence of scattered miliary tubercles in the mucous and submucous coats. Their structure is typical (*Fig.* 444), with follicular arrangement of the epithelioid cells and large central giant cells with peripherally arranged nuclei. They have

FIG. 443.—*Case* 1. Section through the ulcer, stained by Masson's trichromic method; muscle red, collagen green, mucosa purple. The chronicity of the ulcerative process is indicated by the amount and density of the fibrosis in floor and margins. ($\times \frac{1}{2}$.)

nowhere progressed to central caseation, but one or two of the more superficial show central purulent infiltration. The distribution of the tubercles is widespread throughout the excised portions of the stomach, and they occur at all depths of the mucosa. Everywhere few in number, they are least

abundant in the superficial layers of the submucosa, and none are found below this level. Prolonged search through a series of 40 sections failed to reveal tubercle bacilli. There is no evidence of intestinal heterotopia.

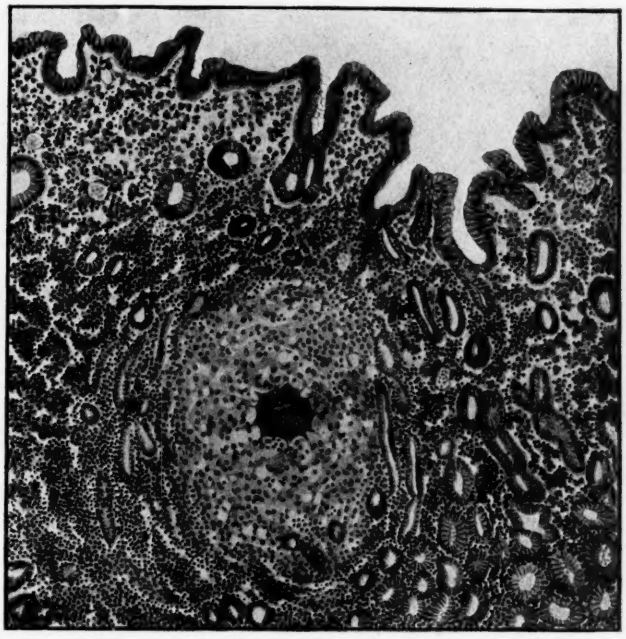

FIG. 444.—*Case* 1. A tubercle follicle in the substance of the gastric mucosa and situated not far from the margin of the peptic ulcer.

Case 2.—*

CLINICAL HISTORY.—The patient, a married woman of 45, gave a twenty years' history of gastric trouble. She had suffered from intermittent attacks of epigastric pain, with intervals of freedom extending to one year. The pain was accompanied by nausea and vomiting, the latter two to three hours after a meal, and she had vomited blood on several occasions. The last attack, which commenced ten weeks before admission, was less severe than some which preceded it. There had been no symptoms indicative of pulmonary disease. On examination there was an area of tenderness in the epigastrium, extending to the right of the middle line. Radiologically there was a filling defect of the pyloric half of the stomach involving chiefly the greater curvature and extending right up to the pyloric ring.

At operation on Feb. 17, 1926, a cicatricial hour-glass stomach was disclosed, with extensive and dense adhesions. Partial gastrectomy was performed, from which the patient made a good recovery. She is now (January, 1928) in excellent health, and altogether enthusiastic about the results of the operation.

* This case was under the care of Mr. Ethelbert Flint, who has very kindly allowed us to incorporate it.

NAKED-EYE EXAMINATION OF SPECIMEN.—Appreciably more than half the stomach has been removed. Externally there is much peritoneal fibrosis and cicatricial contraction on the lesser curvature and adjacent portions of the anterior and posterior walls, with fairly advanced hour-glass contraction

FIG. 445.—*Case* 2. Section of chronic peptic ulcer of stomach situated within ¼ in. of the pylorus. There was completely healed cicatricial hour-glass contraction at a higher level.

a little below the middle of the stomach, and indications of extensive adhesions to adjacent structures. When the organ is laid open, the lesion accounting for the hour-glass contraction and its associated adhesions is found to be a large and densely fibrous cicatrix, apparently completely healed. It is

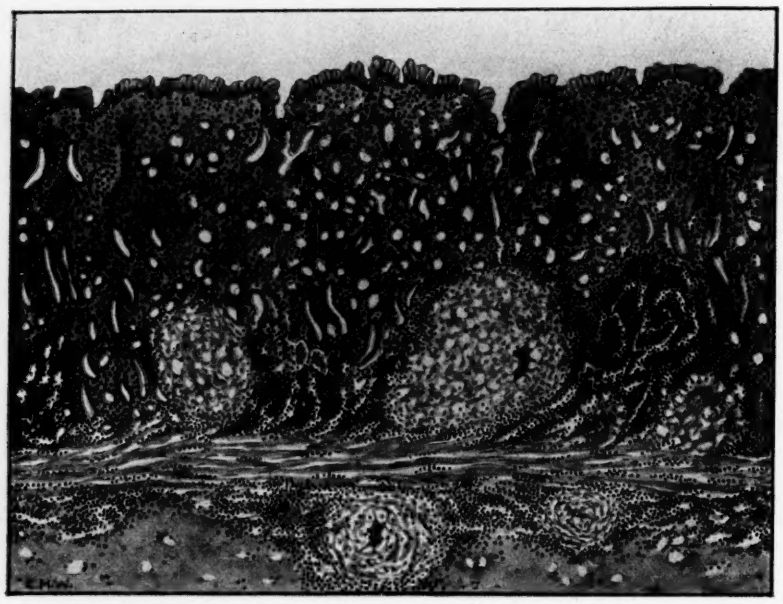

FIG. 446.—*Case* 2. The gastric mucosa at some distance from the ulcer, showing tubercle follicles on both sides of the muscularis mucosæ.

situated astride the lesser curvature at a point 1½ in. from the pylorus, and has caused the circumference of the stomach to be reduced to little more than 2 in. An open chronic ulcer ½ in. in diameter is also present about ¼ in. above the pylorus. The stomach wall here is very œdematous.

HISTOLOGICAL EXAMINATION.—

The chronic ulcer at the pylorus (*Fig.* 445) has the usual histological characters of this lesion. There is an extensive breach of the muscular coat, and the floor and margins of the ulcer are densely fibrosed. There is considerable epithelialization at one margin, and much eosinophil infiltration of the granulation-tissue zone of the floor. The subserosa and adipose tissue of the lesser curvature are the seat of intense vascular engorgement, with scattered hæmorrhages of small size.

The area of scarring 1½ in. from the pylorus is very extensive, and the muscular coat has been breached at two separate points. The mucosa here is of simple regenerative type, and is deficient over a tiny area in the centre of the scar.

As in *Case* 1, the tuberculous lesion, of miliary type (*Fig.* 446), is widely scattered throughout the mucosa of the excised portion of the stomach. It is mainly confined to the deeper layers of the mucous membrane, including a few tubercles just outside the muscularis mucosæ, but one or two are also present in the submucosa. The tubercle follicles are characteristic and well formed, and possess central giant cells with peripherally arranged nuclei. Caseation is minimal, and no tubercle bacilli have been found. Much of the gastric mucosa shows well-marked chronic inflammatory change of a non-specific kind, with considerable lymphoid hyperplasia.

Case 3.—

CLINICAL HISTORY.—The patient, a man of 31 years, began to have attacks of indigestion in 1915. Pain in the epigastrium came on about an hour after food, and was relieved by taking more food. Occasionally there was vomiting, which also brought relief, but there was no hæmatemesis. Quiescent intervals of varying duration occurred between the attacks. At operation in 1919 a chronic ulcer was found on the lesser curvature of the stomach and was removed by cautery (Balfour's method), while the appendix was also excised. No gastro-enterostomy was made. Symptoms recurred within a year, and were similar to those he had had before operation. Shortly afterwards there was hæmatemesis and rather profuse melæna. Again he had intervals of relief, often occurring after a hæmorrhage. In 1924 there was a very severe hæmatemesis. Until May, 1927, in spite of these recurring attacks, he maintained a fairly good state of nutrition and was able to continue his employment. Then, in May and June, he had three severe attacks of hæmatemesis, and when seen on June 27, following the third of these, he was profoundly anæmic. On July 1 a pint of citrated blood was transfused.

At operation on July 5 a very large indurated ulcer was found on the lesser curvature, apparently at the site of the original ulcer. It had penetrated the pancreas posteriorly, with much surrounding induration, and in consequence partial gastrectomy was performed with very great difficulty. Antecolic jejunal anastomosis was effected. After operation a pint of blood was transfused.

The patient had a straightforward convalescence and is now (January, 1928) very well, though still rather anæmic. He has no trouble with his food and has put on considerable weight. The examination of the abdomen

yields negative results. In view of the pathological findings, it must be noted that there was nothing in the history to suggest tuberculosis, nor were there any suspicious physical signs.

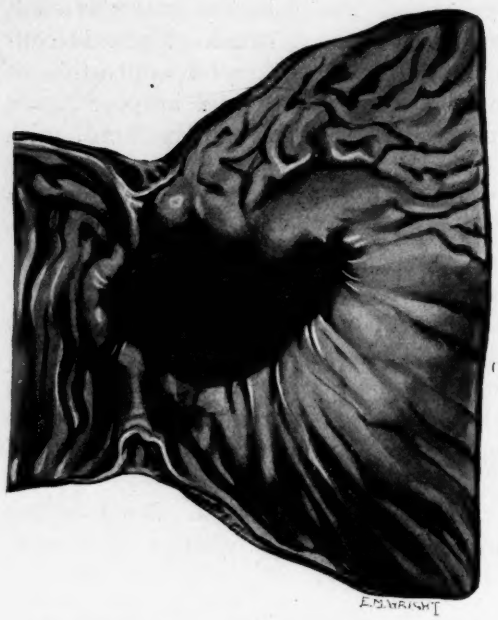

FIG. 447.—*Case* 3. Large 'carcinoma ex ulcere' situated on the lesser curvature and slightly trangressing the pylorus.

NAKED-EYE EXAMINATION OF SPECIMEN.—About half the stomach and fully an inch of the duodenum have been removed. There is a large deep, pyriform ulcer (*Fig.* 447) on the line of the lesser curvature, abutting on, and to a slight extent actually involving, the pylorus. It measures 45 mm. in long diameter, i.e., roughly in the length of the viscus, and 28 mm. across its widest part. The margins are rounded, swollen, and hard, and the floor, which is smooth, is also of firm consistency throughout.

HISTOLOGICAL EXAMINATION.—The microscopic characters are those of a very chronic simple ulcer in which spheroidal-celled carcinoma of scirrhous type is arising in the margins and floor. There is an extensive complete breach of the muscular coat, the floor of the ulcer being formed for the most part of the usual fibrous- and granulation-tissue layers. At the margins there is a very striking fusion of the muscular coat with the muscularis mucosæ. The carcinomatous change commences on the margin of the ulcer at all the five points examined, and extends inwards a variable distance on the cicatrizing floor. In the narrow uppermost part of the ulcer the whole mucosa is restored by tissue which is partly simple regenerative

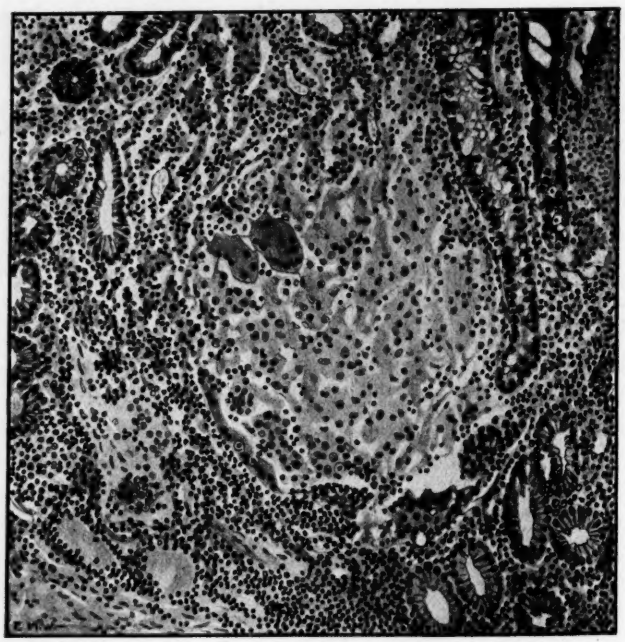

FIG. 448.—*Case* 3. (?) Tubercle follicle in gastric mucosa not far from the margin of the ulcer.

and partly carcinomatous. The malignant changes are characteristically of the type seen in leather-bottle stomach. Affected portions of the mucosa have lost all semblance of their normal structure and consist of loosely aggregated, spheroidal epithelial cells lying in a fine connective-tissue reticulum. Many of the cancer cells, especially those nearer the surface, are swollen, pale, and vesicular from mucoid change. The muscularis mucosæ is more or less continuous, but infiltrated and penetrated at many points by small groups of cancer cells. The submucous and muscular coats are everywhere irregularly infiltrated by carcinoma with much related fibrosis, but the general topography of parts is well preserved. There is much less cancerous invasion of the fibrous floor of the ulcer than of the surrounding tissues. In the mucosa the cancerous change ceases at a short distance from the margin of the ulcer, approximately 5 to 10 mm. At two points in the mucous membrane, each a few millimetres from the edge of the ulcer, there are structures resembling tubercle follicles (*Fig.* 448), aggregations of epithelioid-like cells, with several multinucleated giant cells of Langhans' type and a few polymorph leucocytes centrally situated. Again, no tubercle bacilli have been demonstrated.

COMMENTARY

The cases here recorded are examples of dual pathology of a kind rarely met with. In *Cases* 1 and 2 each of the lesions present is typical, and there is apparently nothing to connect one with the other. It is obvious, both from the clinical histories and the anatomical findings, that the peptic lesions are, in each case, of very long standing. The miliary tuberculosis of the mucosa, on the other hand, is equally clearly the result of recent infection. So far as is known, no other tuberculous lesion was present in either patient, but too much stress should not be placed upon a negative observation of this kind. On the other hand, the good general condition of the patients and their excellent recovery from very severe operations argue strongly against the possibility of the stomach lesion being part of a general miliary tuberculosis. There remains, then, the possibility of a direct infection of the gastric mucosa, either by tubercle bacilli ingested from without or, alternatively, by swallowed infected sputum from some cryptic focus in the respiratory tract. Of these the latter seems the more likely. In passing, it may be remarked that most of the experimental work which has been done on the subject (Broders, 1917) goes to show that gastric juice has little or no effect on the tubercle bacillus unless the contact extends over a period of at least twelve hours. *Case* 3 differs from the others, first, in that the peptic ulcer has undergone malignant metamorphosis, and secondly, that the granulomatous foci, extremely few in number, are not so unmistakably tuberculous histologically.

In none of the three have tubercle bacilli been demonstrated microscopically in the lesions, and to that extent the final and complete proof of tuberculous infection is lacking. The highly characteristic histological picture in two of the cases, on the other hand, must be regarded as very strong evidence indeed. Hurst[3] (1925) has recently described a case of associated chronic peptic ulcer and miliary tuberculosis of the stomach which, clinically

and pathologically, bears a close resemblance to *Case* 1 of the present series. The X-ray appearance of the chest, however, suggested the presence of chronic pulmonary tuberculous disease, and the author considers it probable that the lesion of the stomach was caused by secondary infection of a chronic gastric ulcer with tubercle bacilli from swallowed sputum. The difficulty of distinguishing from malignant degeneration of a chronic ulcer is pointed out.

That gastric ulcer has a tuberculous etiology (Kodon,[4] 1910) is a theory which has never received much support and which is now entirely discredited (Hauser,[5] 1926). In this connection we have investigated in the post-mortem room the general incidence of active tuberculosis in cases of peptic lesions of the stomach and duodenum as contrasted with the incidence in a series of 1000 consecutive unselected autopsies. The results are shown in the table below.

INCIDENCE OF ACTIVE TUBERCULOSIS.

	No. of Cases of Tuberculosis	Percentage Incidence	Difference in Percentage Incidence	Standard Error of Difference
In 1000 consecutive autopsies ..	86	8·6	—	—
In 151 cases of acute gastric and duodenal ulcer	5	3·4	5·2	1·7
In 282 cases of chronic ulcer or scarring of stomach and duodenum	8	2·1	6·5	1·2

From these figures it would appear that the association of the two lesions in the cases just reported is accidental, the infrequency being dependent, in the main, on the great rarity of tuberculous infection of the stomach. Instead of a positive the figures actually show a negative relationship—in other words, the type of person who develops a gastric or duodenal ulcer is less commonly the subject of tuberculous infection than the general population.

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'PAGET'S DISEASE' OF THE GLANS PENIS.

By M. P. SUSMAN, Sydney, Australia.

There are many synonyms for the disorder now generally known as Paget's disease of the skin, e.g., 'malignant papillary dermatitis', 'dermatitis maligna', 'malignant papillitis', epithelioma epidermica', 'eczema epitheliomatosa', 'maladie de Paget', 'Pagetsche Krankheit'. Paget[1] based his classical account of the condition on a series of 15 female cases in which the nipple was affected; but even at this time he knew that the disease was not peculiar to this site. He says: "I believe that a nearly similar sequence of events may be observed in other parts. I have seen a persistent rawness of the glans penis, like a long-enduring balanitis, followed after more than a year's duration by cancer of the substance of the glans. A chronic soreness or irritation on the surface of the lower lip often long precedes cancer in its substance." While the nipple is the commonest site, a number of cases of the disease in other situations have been recorded, the external genital region being most frequently affected. The following is a list of the 33 extra-mammary cases so far reported: —

Summary of Extra-mammary Cases.

Nose - - - - - 4 Cases
Ravogli,[2] Matzenauer,[3] Stelwagon,[4] Boot[32]

Lip - - - - - 1 Case
Winfield[5]

Neck - - - - - 1 Case
Morris[6]

Axilla - - - - 4 Cases
Holzknecht,[7] Zieler,[8] Satani,[9] Jungmann and Pollitzer[10]

Scapular region - - 1 Case
Winfield[11]

Forearm - - - - 1 Case
Hartzell[12]

Back - - - - - 3 Cases
Ormsby,[13] Towle,[14] Beiot quoted by Sekiguchi[15]

Abdominal wall - - 1 Case
Sheild[16]

Umbilicus - - - - 2 Cases
Fox and Macleod,[17] Milligan[18]

Gluteal region - - - 1 Case
Fordyce[19]

Anus and perineum - - 1 Case
Darier and Couillard[20]

Vulva - - - - - 4 Cases
Dubreuilh,[21] Rosenberg,[22] Grinchar,[23] Kren quoted by Sekiguchi[15]

Scrotum - - - - - 3 Cases
Crocker,[24] Sekiguchi,[15] Davis quoted by Hartzell[12]

Glans penis - - - 6 Cases
Paget,[1] Tarnowsky,[25], Tommasoli,[26] Pick,[27] Sequeira,[28] Davis[29]

I shall briefly review the last group of cases, in which the glans penis is affected, before describing my own case. I can find nothing about Paget's[1] case beyond the incidental reference in the above quotation. Tarnowsky's[25] is inadequately reported; the lesion was at first considered to be syphilitic; no associated cancer was found.

TOMMASOLI.[26]—This patient was a fat man of 66 who had never had venereal disease or any serious illness. He first noticed a discharge from beneath the prepuce and difficulty in retracting the foreskin. On examination a red, dry area was found on the prepuce and a red, moist, shiny lesion on the glans. The condition was treated as balanitis, but it gradually became worse; desquamation occurred and the raw red area was tender. Soon all the mucosa of the glans and prepuce was involved except the urinary meatus. [By contrast, the condition began around the meatus in my case.] A dorsal slit was now made. Later, small superficial erosions appeared, with bright, shiny red bases and sharp margins, but no elevation or induration. The inguinal glands were not affected, the urine was normal, and the other systems were clear. Two years after the onset the prepuce was removed for examination, and the histological appearance was described as 'incipient epithelioma'. The operation wound did not heal well at one spot; an ulcer developed here and gradually spread. Unexpected, but only temporary, improvement followed a 'sea-water cure'; after a few days the condition relapsed and became definitely ulcerated. On the base of the ulcer appeared small red nodules, and the meatus soon became involved. The penis was now amputated close to the pubes and the patient remained well for eight or nine months. Then a malignant growth appeared on the urethral mucosa, and the patient died of uræmia with a typical spreading carcinoma.

PICK.[27]—When first seen this man had an 'eczema of the glans penis' of one and a half years' duration; there was a weeping raw surface, and later a proliferation of the epithelium and the formation of nodules. Operation was done for the associated phimosis, and healing occurred by first intention. The condition improved for a time, but soon relapsed and spread widely. Biopsy showed small-cell infiltration and psorosperms (Paget's cells). Pick does not give the end-result of this case, nor say whether cancer was present or not.

DAVIS.[29]—This patient was 70 years old and in good general health. Two years previously he had noticed a small eczema-like patch at the urinary meatus; this spread slowly but caused no inconvenience. The lesion involved one-half of the glans, and was of irregular shape with a sharp margin; the surface was glazed, raw, slightly tender, and had a mucoid discharge; there were two small nodules near the meatus. The inguinal glands were not involved. No biopsy was made and the end-result is not given.

SEQUEIRA.[28]—Sequeira's patient was a commercial traveller, age 77. He first noticed a painful, red, weeping patch at the junction of prepuce and glans two years before. There was no infiltration and the inguinal glands were not involved. Local applications failed, but radiotherapy caused temporary improvement. Later, however, the lesion spread and the glans penis was removed by operation. The patient died of uræmia; at autopsy a round nodule of cancer was found in the bulbous urethra.

AUTHOR'S CASE.

J. S. a painter, aged 60, complained amongst other things of an itchy excoriation around the urethral meatus of a year's duration; he said he had difficulty in urination because the edges of the orifice stuck together at times. He was an unhealthy-looking man, with evidence of visceral syphilis, including aortic disease and Argyll Robertson pupils. The urine was normal. The inguinal glands were shotty but not enlarged. The lesion on the glans is

well shown in *Fig.* 449, and if any description is necessary, I cannot do better than quote again from Paget's original paper: "There is a florid, intensely red, raw surface, very finely granular, as if nearly the whole thickness of the epidermis were removed, like the surface of very acute eczema or like that of an acute balanitis . . . there was always clear, yellowish, viscid exudation. . . . The sensations were commonly tingling, itching, and burning, but the malady was never attended by disturbance of health. . . . The eruption has resisted all treatment local and general." To this account I would add two features that were present in my case, and have been noticed by others—namely, the sharp definition and the slight induration, points which distinguish it from ordinary eczema. I found no deep carcinoma, and I amputated the penis just in front of the pubes. So far (three months after operation) the local condition is satisfactory.

FIG. 449.—'Paget's disease' of the glans penis.

Histology.—The microscopical appearances in this penile case are similar to those seen in the usual breast case. The normal epithelium is seen to pass abruptly into a thickened patch (*Fig.* 450); the thickness is due to proliferation of the Malpighian layer, and it extends into the corium. There is general round-cell infiltration.

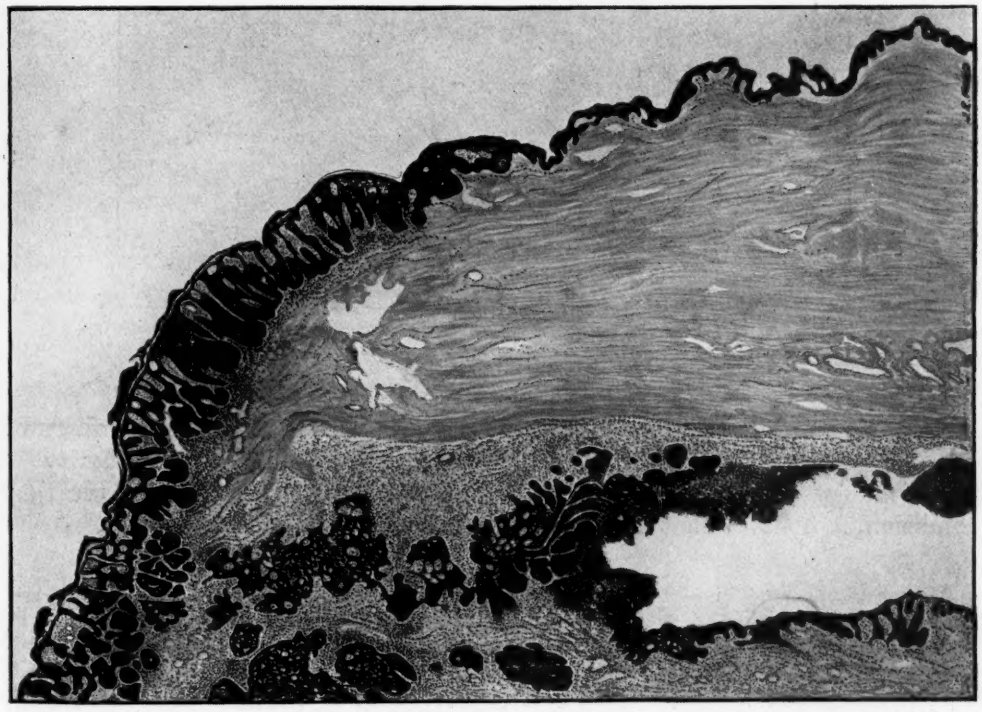

FIG. 450.—Low-power view, including margin of diseased area.

The clearer areas in the thickened epithelium represent degenerate cells or groups of cells. Under high power (*Fig.* 451) the characteristic Paget cells are seen; these are round bodies larger than normal epidermal cells, with homogeneous cytoplasm and deeply staining nuclei. After careful search, several undergoing mitosis were found. These cells were regarded as parasites (coccidia) at one time, but it is now generally agreed that they are derived from the prickle-cells of the epithelium. In the corium there is round-cell

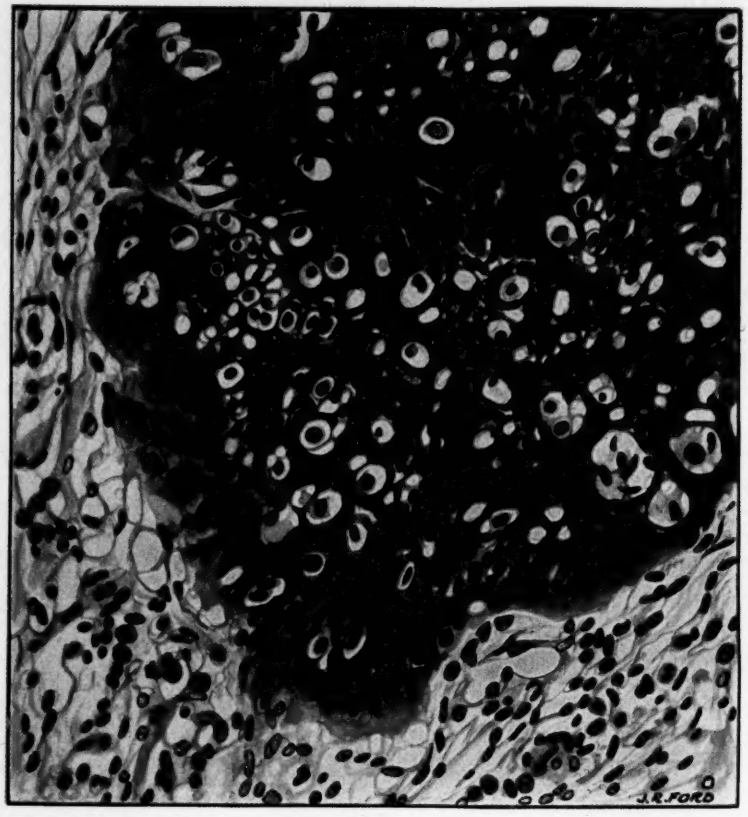

FIG. 451.—High-power view, showing Paget cells.

infiltration and hyperplasia of the connective tissue, where this is not obscured by the proliferating epithelium. I would stress the general similarity of the histology of this lesion to that of basal-cell carcinoma (rodent ulcer), a condition, I believe, that has never been seen on the glans penis.

COMMENTARY.

Relation of Paget's Disease to Malignant Disease.—There is no recorded case of advanced Paget's disease of the nipple that has not been accompanied by cancer of the breast. Macleod[34] says that in non-breast cases also there is an underlying cancer. Cheatle,[30] however, states that extra-mammary

cases are not always associated with cancer, but he gives no details. Of the penile cases, Paget's ended with carcinoma of the glans, Sequeira's with carcinoma of the bulbous urethra, and Tommasoli's with carcinoma in the stump of the urethra eight or nine months after amputation of the penis at the pubes. Tarnowsky says definitely that there was no cancer in his case. It is not mentioned in the other two (Pick's and Davis's). I found no sign of underlying cancer in my patient. Of the remaining extra-mammary cases, eight were definitely accompanied, in time, by carcinoma—as reported by Ravogli,[2] Winfield,[5] Satani,[9] Jungmann and Pollitzer,[10] Hartzell,[12] Crocker,[24] Rolleston and Hunt[33] (2 cases).

The histological similarity to rodent ulcer noted above has also been observed by Cheatle,[30] Sekiguchi,[15] Fordyce,[19] Crocker,[24] and Rolleston and Hunt.[33] Clinically the two conditions are dissimilar, though Paget[1] wrote that "once have I seen it [Paget's disease] pass into a deeper ulceration of the skin after the manner of a rodent ulcer." No case of rodent ulcer of the glans penis has yet been recorded.

Differential Diagnosis.—

Syphilis.—The erosive type of chancre may cause doubt, Paget's disease being mistaken for this rather than the chancre for Paget's disease. A tertiary syphilitic eruption may resemble Paget's disease, and was considered in my patient, who is definitely syphilitic; but the histological appearances are decisive.

Lichen Planus.—This has a dry and unbroken, lavender-coloured, and polished surface.

Recurrent Herpes.—The surface in recurrent herpes is usually sodden and dotted with vesicles, and is not so red.

Drug Eruption.—Therapeutic erosions, such as follow careless use of lysol and other antiseptics, may simulate Paget's disease; but they heal as soon as the applications are stopped.

Diabetic Eruption.—There is glycosuria, and when this is controlled the eruption disappears.

The chief points in the diagnosis are: (1) The red, granular, glazed surface; (2) Induration; (3) Definition; (4) Chronicity; (5) The microscopical picture.

Treatment.—It is very doubtful if any case of Paget's disease has ever been cured by local treatment. Stelwagon[4] mentions four cases cured by X rays and one by radium; others, including Sequeira,[28] have regretted wasting time by trying radiotherapy, for, although the skin lesion vanished in some instances, carcinoma appeared subsequently. The only safe and rational treatment is surgical removal as early as possible.

SUMMARY.

1. Paget's disease of the nipple is fairly common, but only 34 extra-mammary cases have been reported; the external genital region was affected in 14 of these, the glans penis being the site in 7 (counting the present case). One penile case was seen by Paget over fifty years ago; this suggests that the condition may be more common than we think, but either is overlooked or not seen till obvious cancer is present.

2. The clinical and histological features are practically the same whatever the site of the disease.

3. The condition is essentially malignant. At present, therefore, the only rational treatment is surgical.

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CELLULAR HYALINE TRANSFORMATION AND CALCIFICATION IN A SUBGLUTEAL LIPOMA.

By Sir CHARLES GORDON-WATSON, K.B.E., C.M.G.,

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THE following case seems worthy of record because of the remarkable microscopic appearances of the tumour, appearances which make the specimen extremely rare, if not unique, and because clinically and radiographically the case was mistaken for an ossifying sarcoma.

HISTORY.—In May, 1925, the patient, a girl, age 20, first noticed a swelling on the outer side of the right hip, which, from her description, was in close relation with the great trochanter. The swelling gradually increased in size, but caused no pain and no interference with the movements of the lower extremity. On Oct. 12 the patient went to Ryde Hospital owing to the large size which the swelling had attained. She was under observation for five days, and after a skiagram had been taken was discharged as suffering from inoperable sarcoma. Later on her doctor sent her to St. Bartholomew's Hospital, where she was admitted on Jan. 1, 1926. The patient remembers having fallen on to the right hip at the age of 13—seven years ago—a fact which she recalled immediately she noticed the swelling complained of. She was laid up for a month with pain and inability to walk. The swelling seemed to be on the bone, but no bruising was seen. There was no history of a more recent trauma or of disease, and no loss of weight.

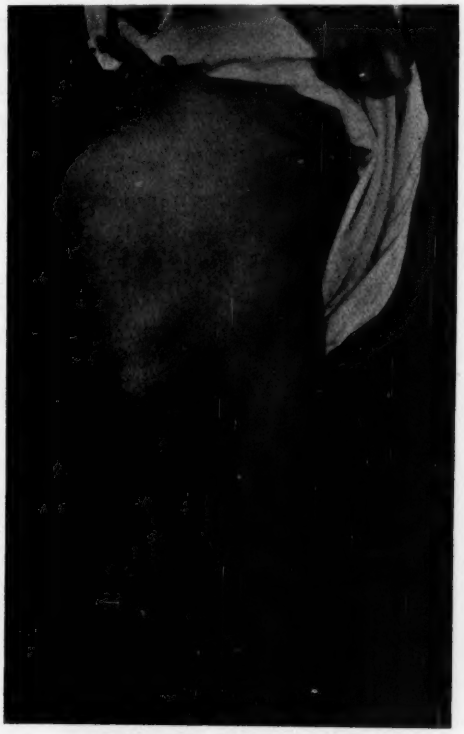

Fig. 452.—Appearance of tumour on admission.

ON ADMISSION.—The patient is a healthy-looking girl, with a postural scoliosis. Over the outer surface of the ilium and upper part of the right thigh there is a large oval swelling which extends downwards from just below the iliac crest for a distance of 12 in. and is 18 in. in its widest part (*Fig.* 452). The skin over it is normal. The tumour is lobulated and smooth, and the skin moves over it. The consistency varies from

a solid nodular firmness above to a soft and fluctuating area below. The upper part of the tumour is fixed to the deeper structures, but there is some mobility of the lower extremity of the tumour, and the femur can be rotated without affecting the swelling, which appears to be attached to the ilium. Movement at the hip-joint is free, and only a slight obstruction to the general movement of the extremity at this joint is noted, which is attributed to the mechanical obstruction of the swelling.

X-Ray Report.—"Films show a very large tumour mass in the upper part of the right thigh posteriorly, with shadows of varying densities throughout it. The appearance suggests a chondrosarcoma, but against this is the fact that the outline of the normal bones seems to be everywhere intact. An alternative that suggests itself is that the appearance is due to a large caseating abscess." (*Fig.* 453.)

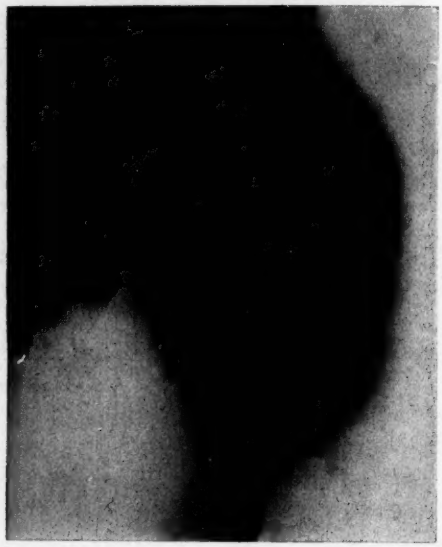

Fig. 453.—Radiographic appearance of tumour.

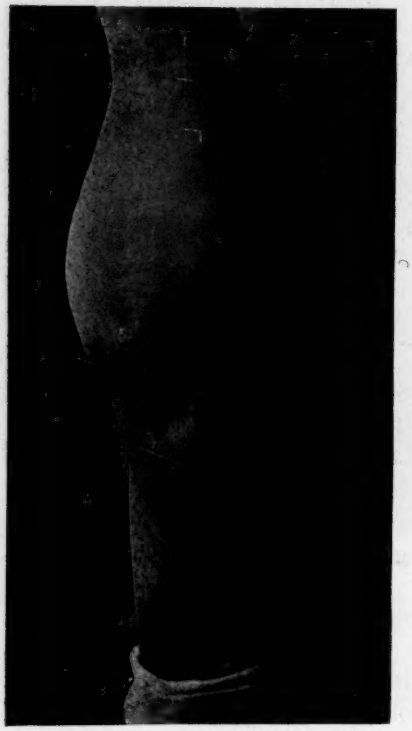

Fig. 454.—Appearance of patient after operation.

The case was shown at surgical consultations at St. Bartholomew's, and the general opinion was that the swelling was a chondosarcoma in connection with the ilium. The balance of opinion was in favour of removal of the mass as far as possible, followed by deep X-ray treatment. Ligation of the internal iliac artery was recommended as a preliminary step.

Operation (Jan. 25, 1926).—The internal iliac artery was ligatured and the tumour explored. It proved to be an encapsuled, hard, caseous, and calcareous mass beneath the gluteus maximus, not attached to the bone but closely adherent to the muscles. This was removed intact except at the lower end, where the softer portion gave way in places and soft caseous-like material came out. The mass weighed 3½ lb., and in places was stony hard.

On naked-eye examination a diagnosis of tuberculous bursitis with caseation and calcification was suspected, but a microscopic examination left no doubt that the

tumour was a lipoma showing very remarkable hyaline transformation of the fat-cells as well as calcification. *Fig.* 454 shows the appearance of the patient after operation.

I am greatly indebted to Mr. Beadles and Mr. Lawrence, who expended much time and trouble at the Royal College of Surgeons in the investigation and preparation of the microscopic sections.

DESCRIPTION OF THE SPECIMEN.

The following is the description of the specimen (*Fig.* 455), which has been prepared and mounted in the Museum of the Royal College of Surgeons by Mr. C. F. Beadles :—

"An elongated oval tumour measuring 8 in. long which was removed by operation from the buttock. At its upper pole about ½ in. of its thickness has the usual appearance of a lipoma. In the rest of its extent it has undergone degeneration and calcification, consisting of opaque white substance resembling caseous material, with indications of a lobular structure in the form of thick fibrous septa passing into its substance from the surface. Externally the growth is coarsely lobulated and is partly covered by adherent muscular tissue.

"Microscopic examination reveals a well-marked lobulation, the tumour

Fig. 455.—Section of specimen removed at operation. (× ¾.)

substance being divided into distinct areas by fibrous septa. Immediately within the septa there is, in most lobules, a narrow layer of polyhedral cells, probably of the nature of proliferating fat-cells. Within this layer the main mass of the lobules consists of cells which have undergone hyaline degeneration, apparently before the deposition of fat, since no adult fat-cells are present. The degenerated cells are swollen, regularly oval in form, consist of a clear homogeneous substance, and contain a small group of granules in their centres; treated with acid the character of the cells remains unaltered, except that the central granules disappear and in a few instances a nucleus becomes visible. The degenerated cells are separated by a small amount of granular matter exhibiting calcareous changes, and probably resulting from liquefactive degeneration of cells which have escaped hyaline degeneration; otherwise they are completely free and isolated. In some places the individual cells appear to have coalesced and form irregular masses of considerable size. The upper extremity of the growth has the structure of a lipoma."

NOTES ON THE MICROSCOPIC SECTIONS BY MR. LAWRENCE.

(*Figs.* 456, 457.)

It is hardly to be expected, with a tumour exhibiting so peculiar a structure, that the diagnosis of lipoma will be accepted without discussion. The tumour consists of a small upper portion composed of adipose tissue, and a large lower portion having apparently a totally different structure. It may be reasonably suggested that the upper portion is merely an adherent piece of subcutaneous fat; or that, if a lipoma, it is an independent growth; or that, if the tumour is to be considered a single growth, it is of a mixed variety, a lipo-endothelioma, for example; or if not that, then a lipoma undergoing sarcomatous transformation. These seem to be the only alternatives to the diagnosis of lipoma. That the fatty portion is part and parcel of the whole tumour is fairly evident from the fact of its rounding off the growth within the continuation of the capsule of the latter, from the absence of outlying portions connected with the fatty tissue around, and from the fact that, microscopically, it is separated from the main part of the tumour by a fibrous septum which resembles in all respects the septa which divide up the rest of the tumour into lobules. Similar reasoning tells against the fatty portion being an independent lipoma.

Assuming, then, that we have to deal with a single tumour, the presence of hyaline bodies immediately suggests a lipo-endothelioma. Against this view there is in the first place the fact that these bodies differ in structure from the so-called psammoma-bodies. The latter arise through the circular compression of a number of proliferating endothelial cells within vessels, as is indicated by their concentric lamination; the former arise from a hyaline change in isolated cells—they are totally different structures. In the second place, although it is common enough to find cell-masses of large size in endotheliomata, it is almost always possible to detect in some parts of the growth the branching columns, arranged more or less definitely in a network, which chiefly characterize those tumours. No such structure was noted in the present instance, although sections were taken from several different

parts. That the tumour is not a lipoma undergoing sarcomatous change seems to be almost conclusively shown by the total absence of any remnants of lipomatous tissue in the supposed sarcomatous areas; further, the tumour is encapsuled and exhibits no evidences whatever of infiltration. At the periphery of the lobules there is a narrow layer of polymorphous cells which might be regarded as sarcoma cells; among them, however, are many which

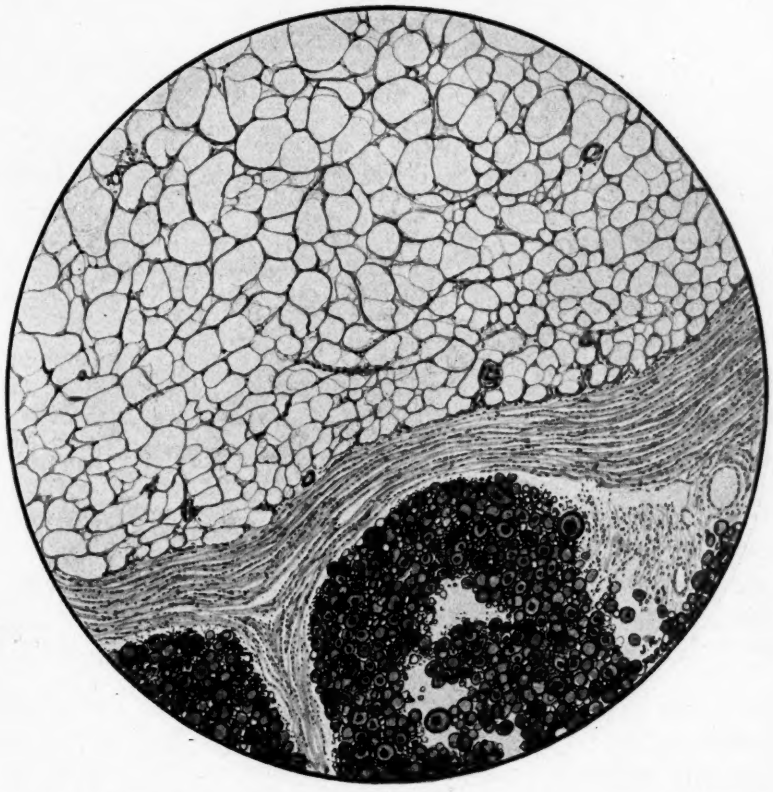

FIG. 456.—Section of the upper part of the tumour, showing parts of three adjacent lobules separated by fibrous septa; the upper lobule consists of lipomatous tissue, the two lower lobules consist of hyaline bodies. (× 50.)

are atypical—in the possession of processes, for example—and there are no multinucleated cells to assist the diagnosis. The subsequent history of the case may throw light on this aspect of the question.

It remains to be shown in what manner the main part of the growth may reasonably be regarded as a modified lipoma. It is known that in rapidly growing lipomata tracts of richly protoplasmic, polymorphous cells occur as precursors of the fully developed fat-cells. We consider that the narrow peripheral layer of cells, already referred to as bounding the periphery of the cell-masses (*Fig.* 457), is composed of cells of this nature. In this early stage such cells contain no fat, and no fat is present in the cell-masses of the growth under discussion. The adoption of this view of the nature of these cells therefore at once involves the diagnosis of lipoma, in spite of the absence

of fat. With regard to the hyaline transformation of the cells, this cannot be looked upon as a degeneration; if it were so it would be impossible to conceive that fully developed, adult fat-cells would not be found in places, whereas there is a complete absence of them. We must suppose that the fat-producing function of the cells has been suppressed, and replaced by the secretion of hyaline as the result of a deranged metabolism.

FIG. 457.—Section of the main part of the tumour, under a high power, showing part of a fibrous septum, adjacent to which is a narrow layer of polymorphous cells, and above this a mass of hyaline bodies. The latter are in some instances clear, in others exhibit remains of a nucleus, in others a collection of granules in their centre. Between the hyaloid bodies are a number of free granules, probably resulting from the breaking-down of cells. (× 340.)

The case is admittedly difficult of interpretation, and so far as we have been able to ascertain from a partial examination of the literature no similar specimen has been recorded.

OSSIFIED POST-RECTAL RECURRENCE FROM A RECTAL CANCER.

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WITH COMMENTS BY SIR CHARLES GORDON-WATSON, K.B.E., C.M.G.,
SURGEON TO ST. BARTHOLOMEW'S AND ST. MARK'S HOSPITALS, LONDON.

THE points of chief interest in the following case are the repeated recurrence in the perineum of a malignant tumour presumably secondary to a rectal cancer five years previously, and the existence of extensive ossification within this recurrence.

The patient, a man, age 53, was first admitted into St. Mark's Hospital in October, 1922, complaining of bleeding from the rectum. A mobile pedunculated adenoma was discovered in the anterolateral wall of the middle third of the rectum, and this was excised by the following technique. The coccyx was removed, the adenoma excised by a posterior proctotomy, the mucous membrane of the rectum oversewn, and the posterior wall of the rectum repaired. The tumour was sectioned, but no evidence of malignancy was found. In November, 1922, the patient returned and sigmoidoscopic examination revealed the presence of two polypi just above the rectosigmoid junction.

In September, 1924, the patient again reported himself with complaint of recurrence of bleeding. Two small adenomata in the lower sigmoid colon were snared, and two others on the posterior wall of the middle third of the rectum avulsed. We have not been able to trace any pathological report on these tumours, but there was no clinical suggestion of malignancy.

In February, 1926, the patient was readmitted, this time under the care of Sir Charles Gordon-Watson, his complaint being a painful swelling in the perineum. Now for the first time there was observed lying underneath the posterior proctotomy scar a hard cystic tumour of about 3 in. diameter. The rectum was free from this mass, and on sigmoidoscopy the mucous membrane was normal.

A transrectus pelvic colostomy was performed as a preliminary to excision. A small nodule was noticed in the right lobe of the liver, and also a hard gland at the bifurcation of the left common iliac artery. Four days after the colostomy, the perineal tumour became painful and tender, the covering skin looked red, and fluctuation was elicited. An abscess was incised and much pus evacuated. On March 9, 1926, the perineal tumour was excised under spinal anæsthesia, together with the 4th and 5th pieces of the sacrum to which the tumour was attached. A portion of the growth remaining in the

3rd piece of the sacrum was treated with diathermy. The wound was left to heal by granulations. Section of the excised tumour showed malignant glandular epithelium invading bone.

In March, 1927, the patient presented himself once more with a perineal tumour at the site of the former operations. His general health was satisfactory; he had not lost weight; his chest was normal; a small ventral hernia was found at the site of the colostomy. The lower border of the liver was just palpable, and was smooth and regular, but apart from this revealed no abnormality. To the left of the broad supple perineal scar a tumour bulged into the perineum and extended laterally to the ischium, superiorly to the remaining portion of the sacrum, and inferiorly to within 1½ in. of the anus. The tumour felt hard: it was narrowly attached to the lower end of the sacrum, but the skin was freely movable over it. The wall of the rectum was normal, and not at all adherent to the postrectal mass.

As a preliminary to a further attempt at excision, the left internal iliac artery was ligatured by the transperitoneal route, after which the patient was placed in the left lateral position and a curved incision 8½ in. long was made, commencing over the 2nd piece of the sacrum and extending downwards along the left natal cleft well to the inner side of the tumour, ending about 1 in. in front of and to the left of the anus. A large flap of skin and subcutaneous tissue was dissected outwards over the left buttock. The underlying lobulated tumour was found attached to the divided sacrum and passing downwards and outwards along the great sacrosciatic ligament as far as the ischium. It was firmly bound to the ligament, but the ischium was not implicated. The mass extended outwards into the substance of the gluteus maximus muscle for about ¾ in. The inner border and lower edge of the tumour was sharply defined and appeared to be definitely encapsulated; so also was the deep aspect. The tumour was dissected from the great sacrosciatic ligament and a portion of this structure removed with it. The mass was then freed on its outer side together with the adherent muscle. Above, the tumour was so firmly incorporated with the 3rd portion of the sacrum that this was excised with it. Further intramedullary spread of the growth into the 2nd piece of the sacrum was curetted and cauterized with pure carbolic acid. During the first stage of the operation, when the abdomen was opened, the abdominal organs were examined, but no evidence of spread was discovered. During the second stage of the operation the fact was confirmed that the rectum and perirectal connective tissue were completely free and normal to all appearances. On recovery from the operation the patient was given a course of deep X rays.

Two months later the patient came to report the presence of a tender swelling at the left side of the lower end of the sacrum, which he attributed to a fall, but no tumour could be seen or felt.

In December, 1927, he returned once more, this time with obvious signs of recurrence. Attached to the lower end of the 2nd piece of the sacrum was a hard mass about the size of a walnut, which was slightly mobile on the sacrum. Painful bony crepitus could be elicited. X-ray examination showed a small ossifying growth in the region of the lower portion of the 2nd piece of the sacrum. This bony tumour, covered by an adventitious bursa, was

cut away from the left extremity of the 2nd piece of the sacrum. An extension of the growth into the sacrum was curetted and cauterized with the actual cautery. The mucous membrane of the rectum was still normal, and the bowel wall was quite free from the growth.

PATHOLOGY.—The macroscopic characters of the large kidney-shaped tumour removed at operation in March, 1927, are well shown in *Fig.* 458. The tissue was very hard, but could be cut with a knife. Many fragments of bone were visible on the cut surface. The portions removed for embedding

FIG. 458.—The tumour removed at the operation in March, 1927, has been opened up to show the extensive ossification found in all regions of the tumour. Note apparent encapsulation. A, Portion of great sacrosciatic ligament; B, Invasion of 3rd piece of sacrum; C, Gluteus maximus.

and sectioning required decalcification. On microscopic examination the tumour was found to be an adenocarcinoma, derived from mucus-secreting columnar epithelium, the supporting stroma of which has undergone extensive calcification. Some of the cancer cells are seen to line cystic spaces containing mucus and cellular débris (*Fig.* 459). Others are packed as solid columns of cells. Although to the naked eye the tumour appears to be enveloped by a compact capsule of fibrous tissue, this is seen on microscopic examination also to be infiltrated by malignant glands. The tumour has all the

appearances of a secondary deposit of a cancer of the large intestine. It has no direct connection with the rectum, and is only narrowly attached to the sacrum.

The bone is found only in the stroma, and particularly at the periphery of the tumour mass. Here the scanty stroma is almost completely composed of lamellæ of compact bone. In other regions, and far removed from the adherent sacral fragment of bone, the stroma consists of a loose cellular connective tissue in the centre of which are spicules of bone. Two types of ossification are to be distinguished in the stroma of the tumour. The commonest is ossification occurring within apparently healthy connective tissue

FIG. 459.—The columnar epithelium is here arranged to form glands and cystic spaces, some of which contain mucus and cellular débris. An irregular fragment of bone is seen to have developed in the loose stroma, exemplifying ossification of living connective tissue. (× 75.)

and not associated with necrosis or calcification. In the other type, exemplified only in one or two places, ossification is taking place within a necrotic cellular mass which previously has undergone calcification.

Commentary.—In view of the information gained by a study of the history of this patient, we have concluded that the so-called adenoma removed from the rectum five years previously must have been an early cancer, and that the post-rectal tumour which subsequently developed and recurred so persistently was a metastasis from this unrecognized rectal cancer.

Perineal secondary deposits of carcinoma of the rectum are comparatively rare, but here we have a tumour of large size growing in muscle and connective tissue and surrounded by a thick capsule of fibrous tissue recurring repeatedly, without undermining the patient's general health and without

apparently any distant dissemination. The very rare feature of ossification may perhaps be accounted for by the fact that the edge of the tumour was attached to the sacrum. The main mass, however, was quite distinct from the sacrum and separated by a fibrous capsule, and active ossification was observed in regions of the perineal tumour several inches distant from the sacrum. New bone was actively growing in the stroma of the tumour, and both the types of ossification which Nicholson has described were recognized—namely, ossification within necrotic and calcified tissue, and ossification proceeding in healthy connective tissue unpreceded by necrosis and calcification. The latter type, comparable to physiological ossification in membrane, was the commonest type observed. We are indebted to Sir Charles Gordon-Watson for permission to report this case.

NOTE BY SIR CHARLES GORDON-WATSON.—This case presents several remarkable features. It seems fairly certain that the papilloma removed in 1922 was an early carcinoma, and that a fragment was implanted in the tissues of the perineum and this slowly developed. The removal of the coccyx was the probable factor in the release of osteoblasts and the formation of an ossifying tumour.

It is the only case that I have met with in which recurrence occurred in the perirectal tissues without involvement of the bowel wall. In the light of subsequent events a colostomy was unnecessary, although at the time of the recurrence it seemed certain that the rectum would have to be excised with the tumour. It is still possible that the colostomy may be closed if further recurrence is avoided.

Another interesting feature in the case is the fact that more than half the sacrum has been removed without upsetting the sacral plexus or giving rise to any peripheral-nerve symptoms.

SOME NOTES ON CARCINOMA OF THE PROSTATE: INCLUDING EVIDENCE OF AN INTRASPINOUS ROUTE OF DISSEMINATION.

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Carcinoma of the prostate shares with malignant disease of the thyroid, suprarenal tissue, and breast the power of giving rise to unexpected and puzzling clinical pictures as a result of distant dissemination. Distant metastases may be the first sign noticeable both to the patient and to the surgeon, and the possible presence of these metastases and their subsequent history often influence detrimentally the surgical treatment. If, for instance, the route of dissemination and the more frequent sites of metastases could be proved to be constant in these malignant growths, treatment of the disease would be more thorough and a better prognosis would therefore result. The work of Sampson Handley and others on carcinoma of the breast has greatly altered the operative and after-treatment of patients suffering from this condition and has given most encouraging results.

The object of these notes is to give, after a few introductory remarks, a brief report of three cases of carcinoma of the prostate to illustrate work of which it is hoped a more detailed report will be published later. It is suggested that, in accordance with the evidence given below, there is, in addition to the other routes, an important intraspinous path of dissemination, and it is hoped that further work will serve to strengthen this theory and possibly assist in the diagnosis and treatment of these cases.

CLINICAL AND HISTOLOGICAL CONSIDERATIONS.

It is not proposed to consider these aspects of the subject in detail.

Clinical.—Carcinoma of the prostate can be divided into three classes: (1) Cases in which prostatic symptoms predominate, viz., urinary obstruction, disturbance of micturition, or hæmaturia; (2) Cases in which pelvic and sacral nerve pain predominate, in many of these urinary symptoms are also present; (3) Cases in which distant dissemination of the growth gives rise to symptoms. In Class 3 the clinical picture may be very variable: pathological fractures; osteoplastic growths of the skull, ribs, or clavicle; pleural irritation; signs of pressure on the central nervous system; or a profound anæmia almost of the pernicious type, may dominate the clinical picture; and, while the presence of a neoplasm is diagnosed, its origin may remain obscure until the autopsy has been performed. In Classes 1 and 2 a routine pelvic examination is usually sufficient to make the diagnosis clear; in Class 3 a rectal examination is often indecisive, although the inguinal glands, as in the other

classes, may be suggestive. Post-mortem examinations suggest that in Class 1 dissemination is not marked in many cases and involves chiefly the local lymph drainage; in Class 2 there is usually widespread dissemination in the pelvic tissues, and the pelvic bones may be extensively involved; in Class 3 the dissemination is often unbelievably widespread. Apart from widespread metastases and renal complications it appears that patients with this condition may live much longer than those with other forms of carcinoma.

Histology.—Examination of the primary and secondary growths has been carried out, and it is not proposed to discuss microscopic findings in any detail in this contribution. It is, however, interesting to note that the cells from a carcinoma of the prostate may exhibit a very marked degree of polymorphism; and this is especially well marked in the distant metastases. The cells growing in loose connective tissue may simulate a round-cell sarcoma and an error of diagnosis result; but if careful search be made in the section, the epithelial nature of the cells can usually be discovered, especially in parts where the connective tissue is more dense. Pelvic lymph-gland deposits often show in the same gland the marked change in cell form and metamorphosis from a definite adenocarcinoma to a more primitive cell form resembling the sarcomata. The cytoplasm alters in quantity and in staining qualities, while the nucleus remains unchanged or may appear to become more distinct, perhaps on account of the contrast with the altered cytoplasm. The grading of these growths in terms of malignancy becomes extremely difficult and uncertain because of this polymorphism; the prostatic growth may show well-differentiated epithelial cells which on Broders' classification would be placed in Grade 1, while the pelvic glands from such a case often show cells which would have to be classed as Grade 3 or Grade 4. Hence any prognosis based on the type of cell taken only from the prostatic growth would be unjustified. In nodules in the liver and some other organs there appears to be very little reaction apart from pressure changes in the neighbouring tissues. The cells in many cases appear to grow along the larger tissue and lymph spaces, and do not appear to have such active infiltrating properties as the malignant cells seen, e.g., in carcinoma of the breast.

REPORT OF CASES.

Case 1.—W. T. B., age 57, admitted for 'general debility and nephritis'. He gave a history of a painless swelling in the middle of the left clavicle which was first noticed two years previous to admission, and which had gradually increased in size. There had been slight frequency of micturition for some months. Five months before admission he had a 'breakdown in health' which he attributed to influenza; at that time he felt extremely weak and became exhausted on the least exertion. He also noticed that his eyelids became 'swollen' and for some weeks had been treated as a case of nephritis, although his blood-urea was only 27 mgrm. per 100 c.c. and no albumin was found in the urine. Seven weeks before admission he felt himself getting much weaker and developed a troublesome cough. On admission he was found to be very anæmic; the head appeared to be somewhat enlarged; and both eyes showed slight proptosis and chemosis. There was a skin nodule about the size of a large pea in the forehead suggestive of a fibrosarcoma; small nodules were also present on the chest. About the middle of the left clavicle there was an irregular swelling about the size of a large walnut, which involved the structures at the root of the neck. The left inguinal glands were enlarged and

stony hard on palpation, while a rectal examination indicated a malignant prostate. He gradually became worse, and died five weeks later. The diagnosis had been confirmed by examination of a section of the excised nodule from the forehead and by a radiogram of the clavicle, which showed an osteoplastic new growth (*Fig.* 460).

POST-MORTEM FINDINGS.—

Head.—The vault of the skull showed osteoplastic deposits of growth (*Fig.* 461) especially marked in the frontal region. In the floor of the anterior fossa there were metastases involving both the orbital plates and the overlying dura; in the left middle fossa there was also a large secondary tumour involving the temporal bone and the dura; no growth was observed in the posterior fossa, and the brain was markedly œdematous but showed no infiltration from the dura.

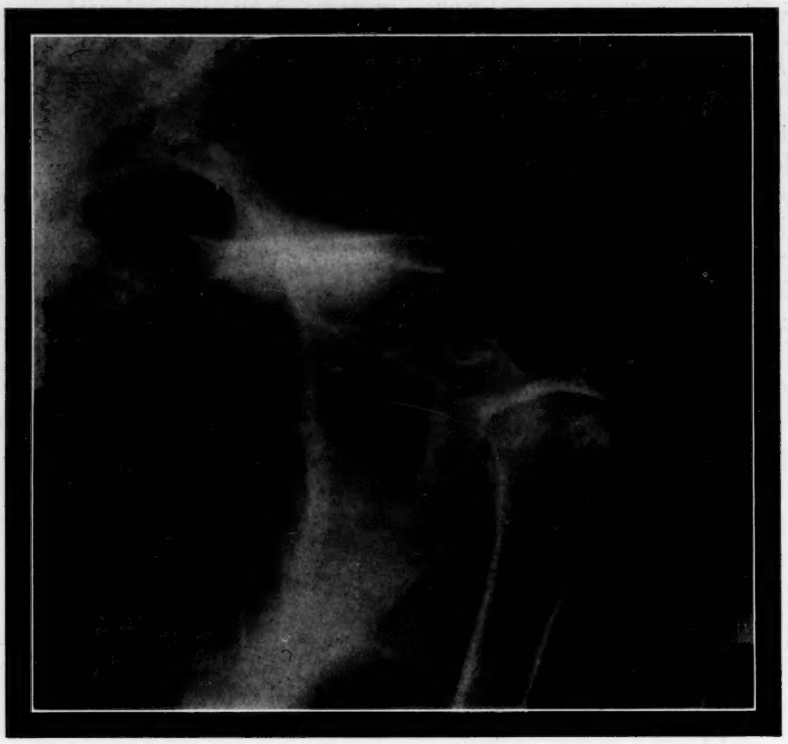

Fig. 460.—*Case* 1. Clavicle. Also shows a pathological fracture through the growth; this occurred two days before the X-ray examination while the patient was reaching over his locker.

Chest.—There was a large nodule apparently arising from the fifth rib on the left side, the overlying pleura being involved and adhesions present along the posterior border of the left lung. The left clavicle was also involved, the growth extending from its posterior attachment to near the sternal end, and involving the tissues in the root of the neck.

Abdomen.—The intestines were distended and the pelvic colon was bound down to the prostatic region; several nodules were present on the surface of the liver. Both kidneys showed chronic nephritic changes; the bladder was trabeculated, and the prostate appeared macroscopically to be infiltrated with a scirrhous carcinoma. The lymph glands along the right internal and common iliac vessels were enlarged, hard, and nodular, and were connected by cords of invaded lymphatic vessels; the left inguinal glands and the external iliac group were also involved.

Before proceeding further, it is important to note the involvement of (1) the skull, (2) the left clavicle, (3) the left fifth rib, (4) the abdominal lymph drainage, macroscopically limited to the pelvic vessels.

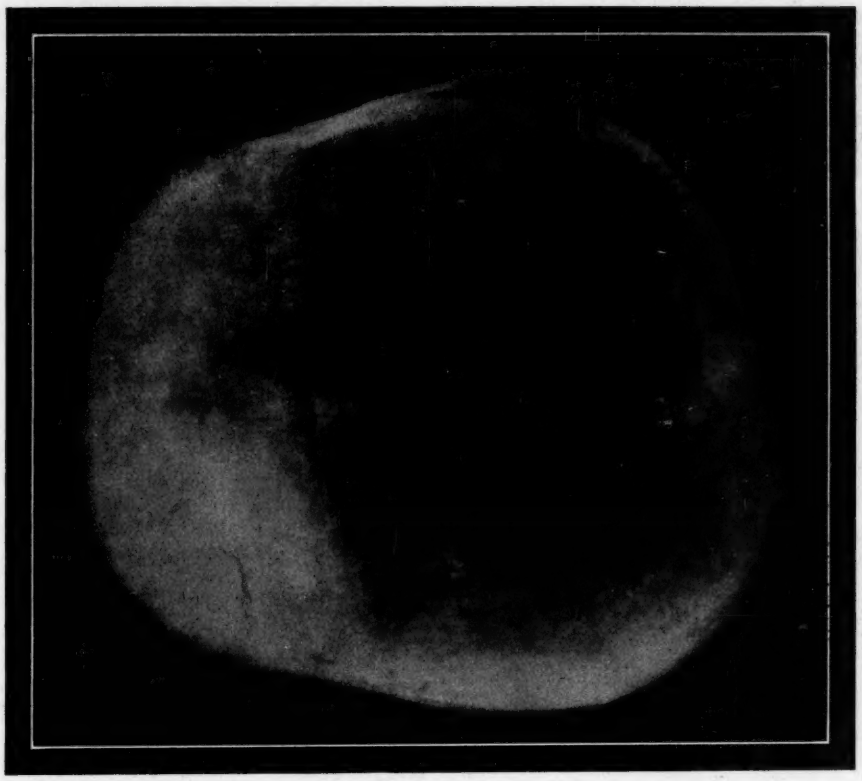

Fig. 461.—*Case* 1. Vault of skull showing extensive metastases.

Case 2.—A. M., age 68, admitted for 'debility and (?) aneurysm'. Apart from a history of gonorrhœal infection fifty years previously, he had been quite well until two months before admission, when he developed left-sided pleurisy, which was followed by constant pain, aggravated by movements, and situated over the sternum, left side of thorax, and back. The patient appeared frail; there was a swelling in the mid-sternum at the level of the third costal cartilage, soft in consistency, having no thrill, no bruit, and being clinically either a gumma or a neoplasm. He complained of marked general weakness, and while being X-rayed sustained a pathological fracture involving the middle of the right femur. Four weeks later he sustained a similar fracture involving the lower end of the left tibia just above the ankle. The size of the swelling increased steadily and the pains increased in intensity until death, which occurred eight weeks after admission.

Post-mortem Findings.—In the head, neck, and thyroid nothing abnormal was discovered.

Chest.—A large tumour mass involved the anterior mediastinum, the sternum, together with the overlying skin. Both lungs were covered with adhesions, and there was a deposit in the hilum of the left lung around the lower bronchus. A large deposit was present on the right side, apparently involving the sides of the bodies of the 2nd, 3rd, and 4th dorsal vertebræ. On the left side there was a deposit involving the posterior part of the fourth rib and the overlying pleura.

Abdomen.—Several nodules with umbilicated centres were seen to be situated on the surface of the liver. There was an aberrant renal artery on either side arising from near the bifurcation of the aorta, and passing behind the ureter to the lower pole of the kidney. Apart from a small nodule on the surface of the right kidney, both kidneys and ureter appeared to be normal. The left suprarenal cortex was enlarged, and on section this appeared to be due to two nodules of growth involving the medulla. The para-aortic lymph glands to the level of the renal vessels were enlarged and hard. The bladder showed little abnormality; but situated in the middle lobe of the prostate there was a nodule which was surrounded by dense fibrous tissue; macroscopically this appeared to be an adenoma, but microscopically the cells were seen to be definitely of a malignant infiltrating type. So far as could be seen the pelvic tissues appeared to be normal.

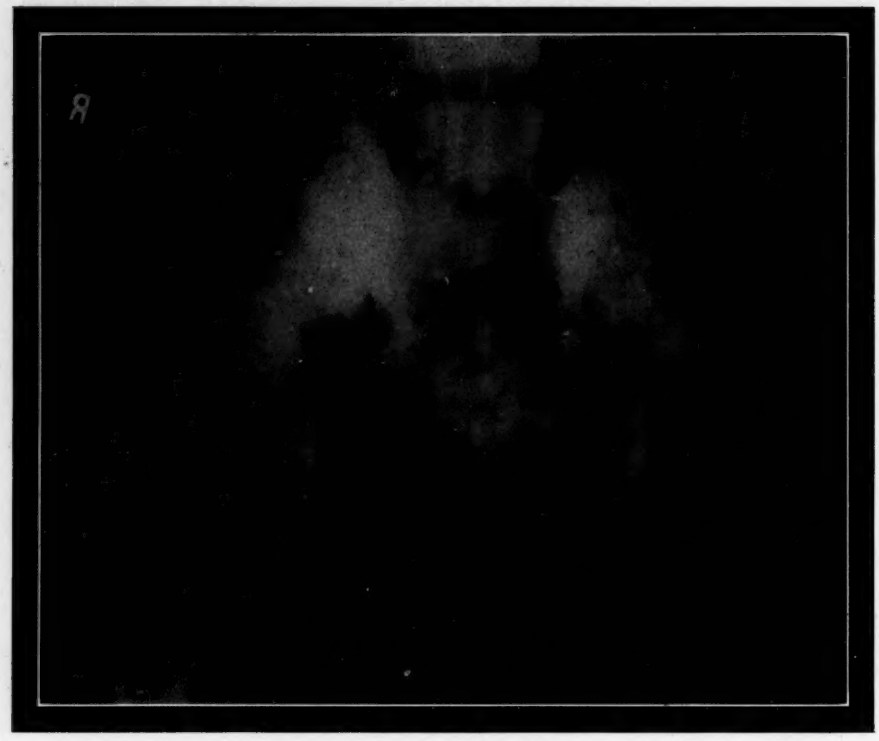

Fig. 462.—Pelvic bones of a case of carcinoma of the prostate. Post-mortem examination refused by relatives.

The outstanding features were, therefore, the involvement of (1) the upper dorsal vertebræ, (2) the fourth left rib—together with the absence of any involvement of the abdominal lymph-vessels above the level of the 2nd lumbar vertebra.

A study of these cases, together with others of a similar nature showing involvement of the skull and upper dorsal region, suggested the possibility of a constant path of dissemination from the primary growth in the prostate. On theoretical considerations the spinal canal was regarded as a possible route, and it was further indicated by the widespread involvement of the pelvic bones sometimes seen in these cases (*Fig.* 462). It was therefore

decided to examine the spinal column in all cases in future, and the following autopsy serves to support the contention.

Case 3.—G. F., age 69, was admitted with the diagnosis of anæmia, marked asthenia, and hæmaturia. Eighteen months previously he had been treated at a general hospital for retention of urine due to carcinoma. He complained of weakness, deafness, and difficulty of micturition. On examination there were hard glands in both groins, and per rectum a fixed infiltrating pelvic growth could be felt. Four weeks after admission he complained of vague pains in the abdomen and left side of the thorax, especially marked over the fourth rib. This was followed a few weeks later by pain in the left arm, the back, and both legs ; and at this time small hard glands could be felt in the right posterior triangle of the neck. He steadily declined, and died five months after admission.

Post-mortem Findings.—

Head.—The vault of the skull showed no evidence of involvement ; in the left anterior fossa, however, there was a nodule which involved the roof of the orbit, while both middle fossæ were involved in deposits which had penetrated the dura, and in addition both temporal bones were extensively infiltrated (*Fig.* 463.) The posterior fossa was clear and the brain apparently normal. The anterior ligaments of the bodies of the 4th and 6th cervical vertebræ were also involved by small plaques of growth.

Fig. 463.—*Case* 3. Left temporal bone.

Chest.—A large metastasis was situated on the left clavicle near its scapular attachment and extending backwards over the first rib. Further deposits involved the posterior ends of the fourth and seventh left ribs, and the overlying pleura was also invaded. Two hard irregular enlarged lymph glands were present on the left side of the aorta at the level of the 4th and 5th dorsal vertebræ, and lymph cords could be seen running from the rib deposits to these glands. The thoracic duct appeared to be quite normal up to the root of the neck. Dense pleural adhesions were situated on the base of the right lung, and both lungs were studded on the visceral pleural surface with small, flat plaques of growth which macroscopically did not appear to invade the underlying tissue.

Abdomen.—The whole of the visceral peritoneum of the small intestines and the colon was studded with little nodules about the size of a large pea, irregular in shape and stony hard. The pelvis was filled with a tumour mass which extended laterally from the prostate and involved both ureters, which were very much dilated. The right kidney showed a pelvic type of hydronephrosis, while the left kidney showed hydronephrosis and also pyelonephritis. The bladder wall was much thickened, and small papilliform nodules were present in the base over the prostate. The lymph glands along the iliac vessels and aorta were all involved as far up as the renal vessels, some of them being as large as a bean; the lymph-vessels could be seen quite distinctly, and in parts formed an almost complete hard encasing network around the aorta. There appeared to be no spread from the aortic lymph-vessels to the anterior ligaments of the vertebræ posteriorly, and the aorta and accompanying permeated lymph casing could be dissected quite freely from the underlying tissues. The spinal laminæ with the ligaments were removed in one piece from the cervical to the lower lumbar region, and at once plaques of growth were seen to involve the *whole length of the ligamenta subflava and the intraspinous surface of*

the laminæ (*Fig.* 464). Both the dura mater and its contents, and the posterior common ligament, appeared to be normal.

The surprising feature was the almost continuous direct spread of growth on the intraspinal surface of the dorsal wall of the spinal canal from the sacral to the cervical region; whereas the abdominal lymph spread had extended only as far as the upper lumbar region. It is interesting to note that *the thoracic duct was uninvolved* throughout its thoracic course. Histological section of the temporal bone showed marked invasion by carcinoma cells of the same type as those found in the pelvic lymph glands and prostate. A section of the lamina in the upper dorsal region also showed columns of carcinoma cells in the tissue spaces of the connective tissue of the ligaments and periosteum of the laminæ; and some of these columns appeared to be surrounded by a layer of endothelial cells suggesting lymph-vessels; others appeared to lie in close contact with the connective-tissue fibres and cells. The spinous process and lamina were also extensively involved.

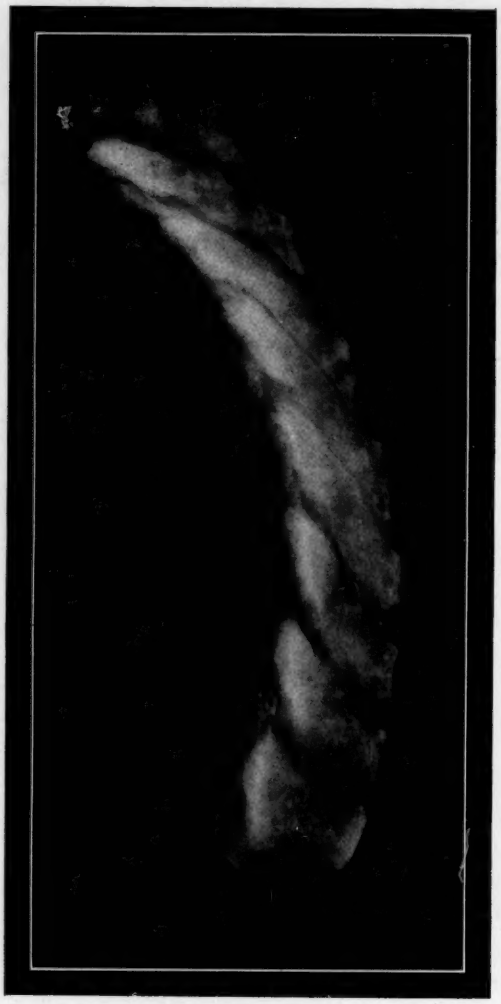

Fig. 464.—*Case* 3. Lateral view of dorsal spinal laminæ.

SUGGESTION AS TO THE PATHS OF SPREAD OF CARCINOMA OF THE PROSTATE.

Malignant growths may spread to other parts of the body by way of the blood-vessels, the lymph-vessels, the serous cavities, or by local infiltration. It is undecided if the blood-vessels play any important rôle in the spread of carcinoma of the prostate. The lower-limb deposits in *Case* 2 were unfortunately not examined with sufficient care to be of scientific value; but the localization of the femoral growth in the middle of the shaft, together with the absence of X-ray changes in the upper end, suggest that the path of spread may have been along the lymph-vessels accompanying the nutrient artery and nerves. Infiltration of local tissues plays an important part in the pelvic symptoms in these cases, and does not differ from infiltration in other forms of new growth. Spread along the normal abdominal lymph drainage of the pelvic organs occurs probably in all cases. The cells from the prostate appear to prefer to travel in the direction of the lymph current, and in most cases do not pass beyond the upper abdominal glands; in one case (not here reported), however, all the para-arterial glands up to the root of the neck

formed a beautiful chain diminishing in size from below upwards. Whether this lymph spread is mainly by permeation or partly by lymph emboli cannot be stated at present. The transcœlomic spread occurs in some cases, and then the peritoneum may be studded with nodules, while those found on the surface of the liver may also result from this mode of spread. In autopsies where the pleuræ have been found to be studded with plaques, metastases in ribs or vertebræ involving the overlying pleura have also been present.

It is suggested, then, that another route plays at least an important (and perhaps the most important) rôle in the distant dissemination of malignant cells from the prostate. This path consists of the lymphatic vessels and tissue spaces of the spinal laminæ with their associated ligaments. Structurally the ligamenta subflava consist of elastic tissue fibres with a network of lymph spaces both between and on the surface of the fibres of which they are composed; also the interspinous ligaments have loose connective tissue between the fibrous framework. The path by which the cells reach the spinal canal is at present unknown, but in a recent post-mortem examination two likely pathways were noted. One path is by the lymph drainage of the prostate to the sacral lymph glands; these glands lie near the anterior sacral foramina and, in this patient, were found to be extensively invaded. It is possible that permeation of lymph-vessels by the carcinomatous cells may obstruct the lymph flow from the sacral glands to the iliac and para-aortic vessels and lead to an increase of flow through the vessels linking up the intraspinal with the intra-abdominal lymph-vessels. Along this path the malignant cell would be able to grow and permeation or emboli occur easily. The other path noted was the loose connective tissue lying on the surface of the levator ani and coccygeus muscles; a tongue of new growth was found passing from the prostate towards the anterior sacral foramina in this tissue. Histologically the growth appeared to be due to infiltration of loose tissue rather than to a lymphatic permeation, but further sections are necessary to decide this question. The pelvic nerves appeared to be free from invasion, as did also the blood-vessels. By this intraspinous route rapid dissemination to distant parts of the body is feasible. The vertebræ, ribs, and skull are accessible to the invading cells, and the involvement of the bone-marrow of these bones may account for the extreme anæmia and asthenia of patients in whom there are such widespread metastases. Theoretically one would expect changes in the lymph-flow in the upper dorsal region, since the main lymph-vessel and its branches are in this region subject to the suction action of the respiratory movements of the thorax and of the negative pressure in the great veins. In this way permeation of vessels in the upper dorsal region, especially of the left side, would be explained.

THE BEARING OF THE PATHS OF DISSEMINATION ON DIAGNOSIS, PROGNOSIS, AND TREATMENT.

Any remarks on this aspect of carcinoma of the prostate are purely theoretical at present.

Diagnosis.—As a rule cases in which prostatic or pelvic symptoms predominate offer no difficulty. In cases where there are distant metastases,

however, the clinical picture is frequently very puzzling, and hence diagnosis is often incorrect. It is suggested that in this group a careful history eliciting such symptoms as backache, vague pains in the limbs, and chest symptoms, together with an examination of the spine including a complete X-ray examination of the dorsal and lumbar vertebræ, as well as of the pelvis, may also assist in the diagnosis.

PROGNOSIS.—Generally speaking the prognosis of carcinoma of the prostate depends on the type of carcinoma cell and on the condition of the kidneys. The majority of cases die as a result of renal failure or of renal infection. A rapidly infiltrating cell may (as in a case recently seen) involve the rectum and peritoneum and lead to a rapidly fatal peritonitis. With good renal function and a slow local dissemination the prognosis depends on the presence or absence of distant metastases and the consequent involvement of the blood-forming organs. It is suggested that if involvement of the spinal laminæ is present the dissemination will be extremely widespread and the prognosis correspondingly unfavourable.

TREATMENT.—In the great majority of cases surgical treatment consists in dealing with urinary obstruction, hæmaturia, or infection—radical treatment being of no avail. Radium inserted into the prostate, prostatic punch or diathermy operations, and permanent cystostomy all have their advocates. From such treatment benefit is frequently derived for many months and even for years in the more slowly-growing types. If, as suggested, the intraspinous path is an important one in dissemination, then the question of deep X-ray therapy or other means of blocking the spinal lymph flow may be considered, and deep radiation of the dorsal and lumbar vertebræ may at least delay, if not prevent, the development of diffuse carcinomatosis.

CONCLUSION.

The suggestion is advanced that there is an intraspinous pathway consisting of the spinal laminæ with their ligaments and the lymph spaces connected with these structures, which *plays a very important rôle* in the dissemination of carcinoma of the prostate, and which can account for the widespread metastases that are not infrequently seen in the upper part of the body in many of these cases.

SPECIAL ARTICLES ON SURGICAL TECHNIQUE.

DIATHERMY OPERATIONS FOR CANCER OF THE TONGUE.

By W. DOUGLAS HARMER,

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In England, the first operation by surgical diathermy was performed in the Throat Department at St. Bartholomew's Hospital by Professor Nagelschmidt, at the invitation of the late Dr. Lewis Jones, in 1910. A vascular nævus of the tongue which had bled freely on many occasions was treated. The onlookers were much impressed by the simplicity of the operation and by the absence of bleeding. The method has been employed constantly since that time for the treatment of malignant ulcers affecting the mucous membranes of the upper air-passages. At first very few surgeons were interested in it, but of recent years the method has become much more popular both in this country and throughout the world. An enormous number of papers have been written, and those who are interested in the subject should consult the books written by Cumberbatch[1] and Wyeth.[2] The former gives admirable descriptions of the different instruments that have been invented, explains in detail the theories of the currents that are used, and clearly lays down rules for the treatment of various surgical conditions. Wyeth's work also contains many new ideas, which have resulted from careful research in many hospitals in America. These books also contain very full references to the papers of surgeons in all parts of the world.

Shortly speaking, diathermy is produced by a current which oscillates 250,000 to 1,000,000 or more times per second. It generates intense heat *within* the tissues and thus coagulates them. If properly applied it is a perfectly safe treatment and produces no general shock—in fact, the nerves and muscles do not react to this type of current. Any form of tissue can be destroyed locally without damage to surrounding parts, and practically without loss of blood. The operations can be completed in a shorter time than with the older methods; there need be no handling of tissues. Afterwards there is rarely any shock; as a rule the temperature is only slightly raised. The absence of pain after this treatment is remarkable owing to the paralysis of the sensory nerves, and as a rule the patients are able to swallow easily within a very short time.

Diathermy is an admirable method of treating cancers of the mucous membranes in any part of the upper air-passages, particularly those that are

easily accessible, such as carcinoma of the tongue. This disease in its primary form is sometimes a very local condition, but it is often difficult to decide how much of the tongue should be removed in order completely to eradicate it. Generally speaking, the growths are very malignant. They tend to disseminate in the glands of the neck early; even small innocent-looking warts may grow with great rapidity.

Years ago I was asked to visit a lunatic asylum and there to examine a doctor who had a small growth on the tongue. There seemed no doubt that this was an innocent papilloma with a smooth surface, about a quarter of an inch in diameter, and a fairly thin definite pedicle. As he was a raving lunatic and did not complain of his tongue, it was decided that the wart should not be removed. Six months later the growth had involved half of the tongue, and there were massive glands on both sides of the neck, with fungations through the skin at two places.

On the other hand, occasionally one meets with cases in which after free removal of the primary growth by some simple operation there is never any recurrence either locally or in the neck. It is therefore very difficult to give a prognosis in a case of cancer of the tongue. Certainly the size and appearance of the primary growth is no guide. In all these ulcers there is a virulent poison—possibly an ultra-microscopic organism—which is constantly exuded from their surface and doubtless is present in great quantity in every part of the mouth. There can be little doubt that when these ulcers are removed with the knife this poison enters the blood-vessels and lymphatics and so reinfects the wound. This is the principal cause of the recurrences that are so frequent.

Theoretically diathermy is a better treatment than the knife because it produces such intense heat that the poison is thoroughly sterilized. Moreover, if care is taken, the whole growth can be completely destroyed by a bloodless operation, so that the vessels and lymphatics are never infected. To obtain good results it is most important to take stringent precautions against sepsis. In every case septic teeth should be removed before the operation. When the ulcer of the tongue is foul, it should be completely coagulated and dried before any attempt is made to remove it.

Anæsthetic.—Except for the treatment of small localized growths, general anæsthesia is advisable, and chloroform and oxygen, or preferably gas-oxygen-chloroform mixture, should be given. Ether must never be used for fear of burning. With local anæsthetics the patient must be warned of the danger of moving, which may easily result in sparking and burning of normal parts.

Preliminary Laryngotomy is rarely necessary, and only when operating on growths at the back of the tongue, so as to prevent the danger of inhalation pneumonia. As a rule this difficulty can be overcome by intratracheal anæsthesia with chloroform.

Preliminary Ligature of the lingual or external carotid arteries is also rarely needed. When the growth is localized in the anterior two-thirds of the tongue I prefer to ligature the lingual artery in the floor of the mouth after excision of the disease. When treating a growth on the dorsum far back, the vessel need not be ligatured unless the disease extends very deeply

into the submaxillary region, and in such a case preliminary ligature of the external carotid and of the lingual and facial should be performed.

METHODS THAT CAN BE EMPLOYED.

Fulguration.—For fulguration a single needle electrode is best. *Thick* sparks are more effective than thin ones. The tissue on which they fall is quickly coagulated, and as the sparks continue to play on the coagulated tissue it is deprived of water and is converted into a shrunken dry mass. With this form of treatment the heat does not penetrate to the deeper tissues more than a few millimetres, and therefore it is in no sense a true diathermy. Fulguration is suitable for the treatment of patches of superficial leukoplakia, especially those which have suspicious sores or a slightly warty surface. In such cases it is quite unnecessary to destroy the deeper tissues. In fact it is doubtful whether removal of larger portions of the tongue has any effect in stopping the progress of this disease. In cases of leukoplakia it is important to destroy the superficial ulcers as early as possible and to repeat this as often as is necessary by local coagulation. With this treatment it may be possible to prevent any serious spread of the disease for many years. The worst case that I have ever seen of this type was a man who had had leukoplakia for many years. At the age of 63 he began to develop suspicious sores, which were easily cured by fulguration. The treatment had to be applied frequently, and after twelve years a small gland had to be removed from the submental region. This was found to be infected both by tubercle and carcinoma. Eventually, sixteen years after the first treatment, metastases appeared on both sides of the neck and he succumbed very quickly.

Coagulation.—The electrode may be a narrow disc or button (*see Fig.* 468), or a single needle or a group of needles. When a strong current is used the tissue in close vicinity with the electrode is coagulated in a very few seconds and is rapidly dried and rendered non-conductive. Sparks then appear, and true diathermy ceases. If, on the other hand, a weak current is used, the tissue coagulates more slowly, drying is delayed, and there is time for the tissue at greater depth to become hot before sparks appear. Thus, in operating under a local anæsthetic the heat and pain that are experienced by the patient may be felt along the neck as low as the clavicles, although there is no sensation in the tongue. It is possible that this heating through of the tissues is partly responsible for the good results that have been obtained by diathermy.

Coagulation is suitable for the smaller ulcers which originate either on the tongue or on the floor of the mouth. Twelve years ago I treated an ulcer which involved the side of the tongue, the floor of the mouth, and the inner aspect of the lower jaw, extending from the frænum 1½ in. backwards, by local coagulation. Six weeks later the ulcer had healed, but there was still indefinite induration in part of the scar, and a second treatment was given. The wound then healed soundly, and the patient has remained perfectly well since. He has a freely movable tongue and speaks naturally. In this and other similar cases the growth has been successfully destroyed by local coagulation, and practically no deformity of the tongue has resulted.

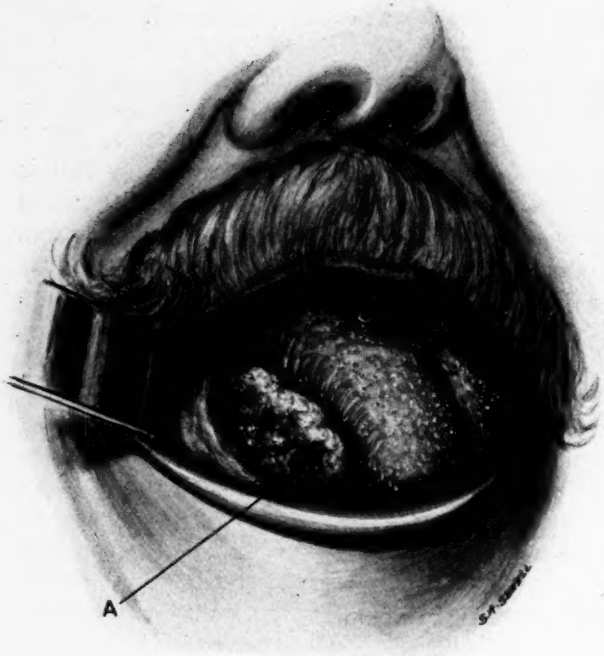

FIG. 465.—The patient received two treatments with diathermy. A, Ulcer of floor of mouth.

Circumvallation.—This treatment was first advocated by Howard Kelly. A fine needle is used to coagulate the healthy tissues around the growth so as to isolate it completely. When half the tongue is to be removed the needle is passed right through from the dorsum to the lower surface, and by repeated punctures the affected part is completely isolated, the vessels and lymphatics being sealed. Using the same needle and current, the ulcer is then completely coagulated so as to sterilize its secretion. With a diathermy knife the affected parts are then excised. Lastly, the wound surface

Moreover, at no period have the glands of the neck required treatment (*Figs.* 465, 466). But this method is only applicable to early growths in the primary stage.

Coagulation followed by Excision.—This was the method that I employed formerly in most of the cases where there was any doubt as to the extent of the involvement of the tongue. Using the button electrode the ulcer is completely destroyed, and then with the diathermy knife the adjacent part of the tongue is freely excised. The method has given good results, but has now been abandoned in favour of the following (*Figs.* 467-469):—

FIG. 466.—The same case as *Fig.* 465. Condition eight years after diathermy. No treatment of the glands.

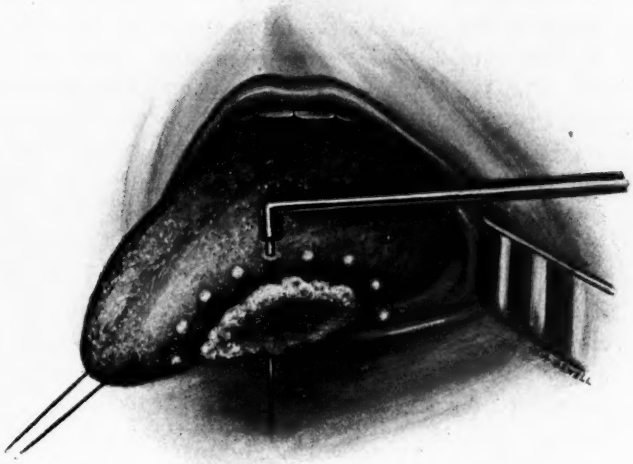

FIG. 467.—First stage. Circumvallation with needles.

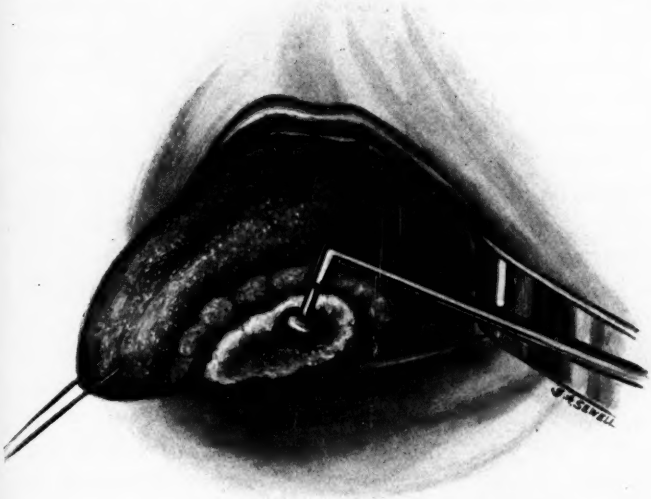

FIG. 468.—Second stage. Coagulation of ulcer with button electrode.

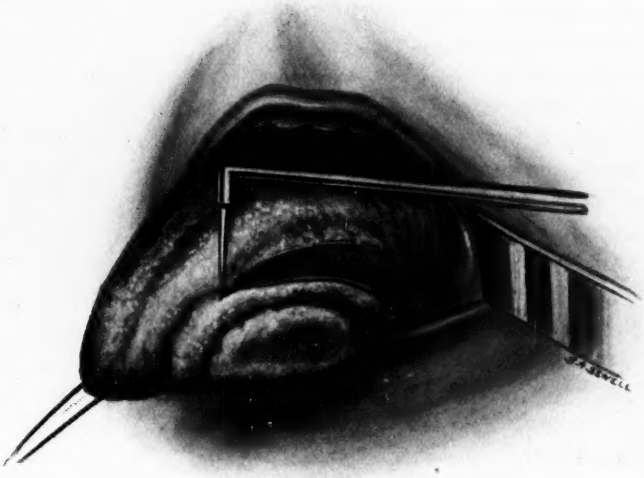

FIG. 469.—Third stage. Endotherm knife—showing excision of the growth.

is seared by fulguration. This is probably the best method that can be adopted for the more serious forms of disease, and it is also the safest way of removing a fragment for microscopical section.

The 'Arc Operation' (Cutting with the Endotherm Knife).—The idea of utilizing high-frequency current for the purpose of cutting the tissues originated with de Forest. Currents which are specially suitable for this purpose are now largely used by Wyeth, Kelly, and Anderson. A needle or narrow blade is used as the active electrode. When it makes contact with the tissues a minute arc appears which surrounds the electrode when the latter is inserted. When the electrode is drawn rapidly through the tissues they are divided or split by the arc. The operator does not cut the tissue, nor does he feel any sensation of resistance as the electrode splits them asunder. If the knife is moved quickly, coagulation will not penetrate further than a fraction of a millimetre, and so little tissue is destroyed that afterwards the wound can be brought together and sutured, healing taking place by first intention. Wyeth uses this method both for the excision of the tongue and for dissections in the neck. With it he is able to take away the glands in close contact to the carotid vessels without damaging their coats in any way. He claims that dissections performed with this method give better results than when done with the knife.

Monopolar Dehydration.—This is a method invented by William L. Clark. One electrode only is used. A current is passed through it which dehydrates the cells which are in immediate contact (electro-desiccation) without causing any burning or coagulation. The affected tissues are powdered and can be rubbed away without destruction of the underlying healthy cells. This method is used for leukoplakia, ulcers, and superficial neoplasms, warts, or moles.

Radium Followed by Diathermy.—The results obtained by Regaud, Bayet, and many others with radium in cancer of the tongue have been so encouraging that in the last three years I have adopted the following method of treatment. Needles containing 1 mgrm. of radium salt are inserted into and around the growth and retained in position for from four and a half to eight days. In some cases the ulcer entirely disappears, but in others it only becomes smaller and more localized. When the resulting scar is examined microscopically it is found to consist of masses of fibrous tissue enclosing small collections of unchanged cancer cells. For this reason, if the growth does not entirely disappear within six weeks it should be excised by diathermy. Eighteen cases have been treated on these lines with good results. A schoolmaster who had an extensive ulceration involving the whole of the left half of the tongue, the floor of the mouth, and crossing the middle line in the anterior third, was treated by this method. Eleven 1-mgrm. needles were buried for 168 hours and the growth rapidly shrank, leaving an indurated scar along the left margin of the tongue only. This was excised by diathermy three months later. The wound healed rapidly, and two years later the patient had a freely movable tongue and a normal voice. He is now able to carry on his work, which would certainly have been impossible if the whole of the original disease had been removed. *Figs.* 470–472 illustrate the method employed.

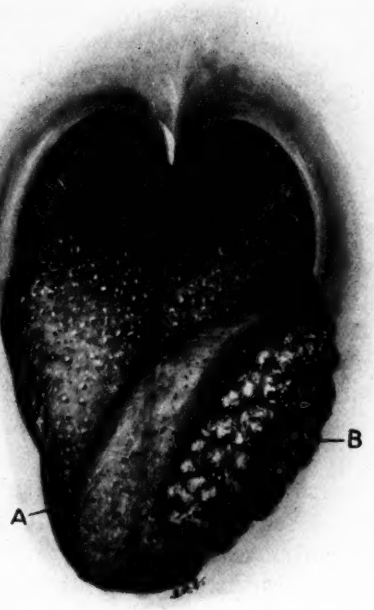

FIG. 470.—Before treatment with radium needles. A, Infiltration; B, Ulcer.

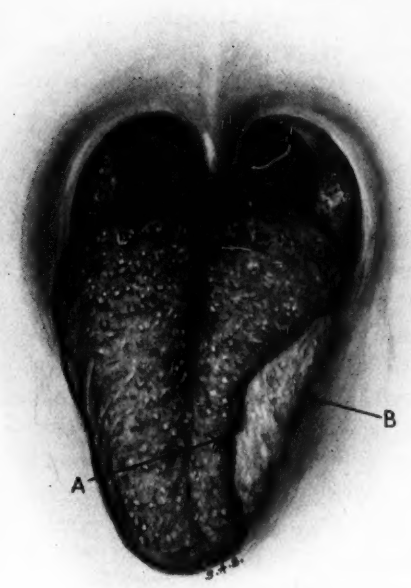

FIG. 471.—The same case as *Fig.* 470. Condition three months after treatment with radium needles. Excision by diathermy. A, Normal tissues; B, Smooth fibrous scar.

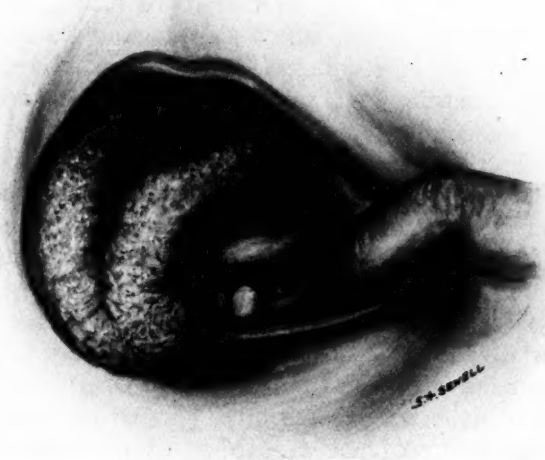

FIG. 472.—The same case as *Fig.* 471, showing a freely movable tongue two years after excision of the scar by diathermy.

At present we have not had sufficient experience of the combined use of radium and diathermy to decide how far it will be useful, but the first results are promising, and if it is found that in most cases the growths can be localized so that it is only necessary to remove a small portion of the tongue, it is obvious that less mutilation will be caused and far better voice results obtained.

HEALING AFTER DIATHERMY.

A few hours after coagulation the parts that have been destroyed are covered with an exudation of lymph, and in a few days by thick membranous sloughs. The latter adhere firmly to the surface, and as a rule do not become septic or necrotic. They begin to separate in about a week, but the process is not usually completed in less than ten to fourteen days. Mouth-washes of Condy's fluid or peroxide of hydrogen should be used frequently. If the sloughs are very offensive, the best treatment is inhalation of oxygen, which causes them to separate rapidly. The atmosphere of the room can be kept sweet by a 'drip' vapour lamp supplied by S. Maw, Son & Sons.

During the stage of sloughing the temperature should not be raised; in fact the absence of toxæmia is remarkable. The tissues under the sloughs remain aseptic, and collections of pus are not formed as under a scab. This accounts for the absence of pain after diathermy. After separation of sloughs rapid epithelization occurs, the whole wound becoming covered with a smooth pliable membrane without formation of scar tissue. The tongue is thus freed and often becomes freely movable—a point of great importance.

Complications are rare. As already stated, sepsis seldom occurs either locally or in the neck if the patient is in good general condition and the disease is not too advanced. But care must be taken not to operate on patients who are very anæmic (cachexia), when the mouth is foul and septic, and when the disease extends too deeply into the tissues of the neck. As a rule it does not benefit the patient merely to coagulate the surface of the growth, except in rare instances where this is done to remove a septic sore or to control hæmorrhage. Secondary hæmorrhage is certainly less common than after cutting operations if suitable cases are treated. Generally it is slight, and occurs in the later periods of the separation of the sloughs, which have become septic. Unless an artery is involved, the bleeding can usually be stopped by simple means, such as an injection of morphia, local pressure, etc. In only two of my cases has the hæmorrhage caused fatal results. In one the whole tongue had been removed without preliminary ligature of the lingual arteries, and the patient died after repeated severe hæmorrhages. In the other case a fairly localized growth involving the base of the tongue and epiglottis was destroyed, and the patient died suddenly after a small hæmorrhage of about four ounces on the tenth day. In this case it is probable that the blood passed into the trachea and asphyxiated the patient.

Œdema of the Floor of the Mouth or of the Neck.—Slight œdema in these situations may occur after prolonged operations, especially if too weak a current is employed and the electrode is left in contact for too long a period, as already explained. As a rule the swelling disappears in a few days, and personally I have never seen any serious inflammation caused by it.

Septic bronchitis or pneumonia is also rarer than formerly, for the simple reason that there is not the same danger of inhalation of blood or septic discharge when the operation is properly conducted. Only two cases of septic infections of the lower air-passages have occurred after this treatment.

TREATMENT OF GLANDS.

With diathermy the treatment of the primary growth is comparatively simple, and good results can be obtained in a high percentage of cases, but the secondary deposits in the glands of the neck are far more difficult to deal with. The presence of metastases in the neck always makes the prognosis unsatisfactory; in fact the chance of a permanent cure when the growth has disseminated is certainly less than half what it is when there is only a primary growth. The type of growth is also important. In the rapidly-growing cellular forms, the so-called Grade 1 types, it is doubtful whether any form of operation on the neck ever gives good results. In fact there is evidence to show that the growth may be stimulated to greater activity. This is not the case in the lower-grade types where the growths are highly keratinized and less malignant. In this connection the work of Broders[3] and others is certainly important, and personally I am becoming more and more convinced that we must pay considerable attention to it. On the other hand, it is well known that the resistance to cancer varies greatly in different patients. Thus, heavy drinking, syphilis, and diseases of the heart, kidneys, etc., often lower the vitality of the tissues generally, and in this class of patients operations are rarely successful. Probably for the same reason patients who have lived in the East for long periods are generally bad subjects.

In deciding the treatment of the glands, therefore, there are many factors which must be considered. Shortly stated it depends on the following conditions :—

1. If no glands can be found, and the growth is not of a virulent type, it is advisable to leave the neck untouched but to examine it periodically for about twelve months to make sure that there is no disease.

2. When dealing with cases of Grade 1 and 2 without glands, the neck should always be treated either with a radium collar or by X rays.

3. If suspicious glands can be felt in various parts of the neck it is more difficult to decide whether to operate or not. When there is doubt as to the extent of the metastases, radium and X rays are generally preferable to an operation.

4. If there is local enlargement of the glands—for instance, in the submaxillary region or on the bifurcation of the carotid—they should always be removed by dissection if possible. As a rule only one side of the neck should be treated. Whenever necessary, a part or the whole of the sternomastoid muscle should be removed. If there is any doubt whether all the disease has been eradicated or not, the patient should afterwards receive full treatment with X rays or radium collar. Levitt is of opinion that the best results from X rays have been obtained in those cases where the greater part of the glands have been previously removed.

5. If there are multiple glands on one or both sides of the neck extending low down towards the clavicles, and especially if they are very adherent to other structures, operations should not be attempted. In some of these cases benefit can be obtained by burying either radium seeds or needles, by applying a radium collar, or by X rays; but each case must be considered individually, especially with regard to expectation of life.

CONCLUSIONS.

Diathermy has now been in use in the treatment of cancer of the tongue for about seventeen years. The results that have been reported by surgeons in all parts of the world are so encouraging that there can be no doubt it is one of the best methods of treatment that has so far been devised. If the operation is properly carried out by the bloodless method there is no danger of disseminating the growth. Certainly it is easier to operate by this means than with the knife. The treatment can be carried out in a shorter time, and there need be no handling of the tissues. Complications are also less frequent than after cutting. Thus there is less shock and toxæmia. The absence of pain is remarkable, and allows of easier swallowing. The sloughing that results is harmless. Many cases which would be considered inoperable by ordinary means can be relieved, if not cured, by the method—in fact, it may be the only means of sterilizing a painful septic sore. Even if life is not prolonged, the existence of the patient may be rendered more bearable.

Possibly by combining radium and diathermy still better results will be obtained and less mutilation produced. The treatment of the glands in the neck still presents the greatest difficulty.

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[2] WYETH, GEORGE A., *Surgery of Neoplastic Diseases by Electrothermic Methods*, 1926. New York: Paul B. Hoeber.
[3] BRODERS, A. C., "Squamous-cell Epithelioma of Lip: a Study of 537 Cases", *Jour. Amer. Med. Assoc.*, 1920, lxxiv, 656.

SHORT NOTES OF RARE OR OBSCURE CASES

CASE OF OSTEOMYELITIS OF RADIUS TREATED BY BONE-GRAFTING.

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This case is of interest in that it shows the gradual development of a good anatomical radius from a bone-graft.

On Nov. 9, 1923, V. L., female, age 3½ years, was admitted to the Hospital for Sick Children, Great Ormond Street, under Mr. Waugh, with a two days' history of pain in the right forearm. Temperature, 100·4°, pulse 144.

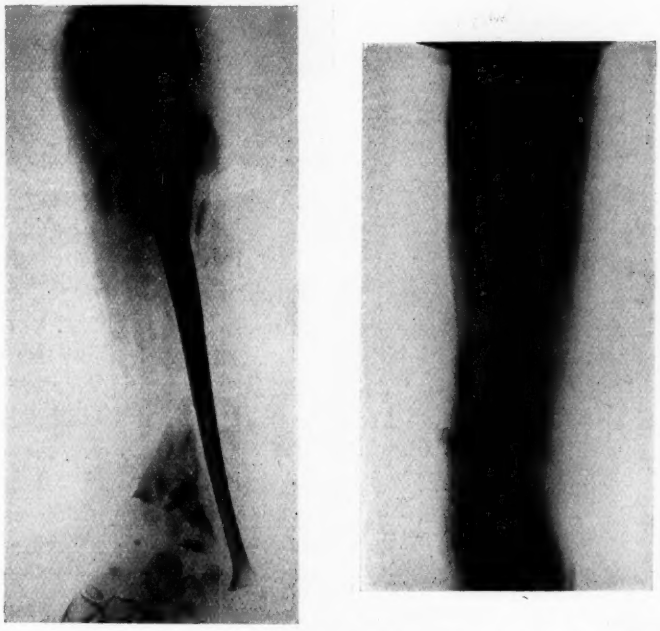


Fig. 473.—Two skiagrams, showing the loss of diaphysis of radius, with small proximal and distal fragments persisting before the insertion of the graft (March 28, 1924.)

On Examination there was general swelling over the whole extensor aspect of the right hand, wrist, and forearm, with some redness and tenderness. There was no definite localized tenderness over either forearm bone, and wrist and elbow-joint movements were full. The swelling and tenderness increased, becoming more localized to the outer aspect, and on Nov. 13 an operation was performed by the author.

FIRST OPERATION.—On exposing the radius along the outer border of the forearm, the periosteum was found lifted from the radius along its entire length, and the bone surrounded by pus. Grey necrotic débris and pus were found in the medulla of the bone, which was guttered along its entire length, the extremities of the bone, as far as possible, not being encroached on. The whole of the infected area of bone was thus thrown open to the surface of the limb in order to carry out the principle of ideal drainage. At the same time the least amount of trauma possible in the presence of such widespread infection was inflicted upon the growing end of the bone at each extremity. This is a necessary provision for subsequent growth in length of a graft, when, as in this case, the need for the inlaying of a graft at a later

FIG. 474.—Bone-graft in position (April 24).

FIG. 475.—Two skiagrams to show the absorption of bone-graft with new bone formation (*a*, June 28, and *b*, Aug. 13).

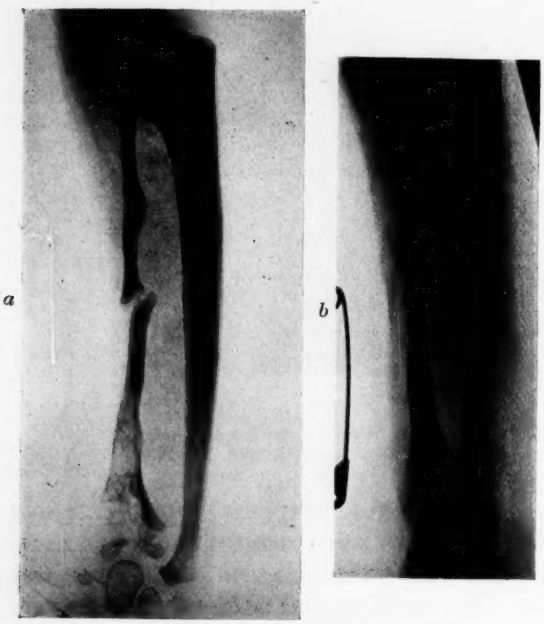


FIG. 476.—Two skiagrams to show the fracture and subsequent union of bone-graft. *a*, Taken on Nov. 3, 1924, and *b*, on Feb. 16, 1925.

date can be foreseen at the original operation. Carrel tubes were inserted, and intermittent irrigation with ½ per cent chloramine-T solution was begun. *Staphylococcus aureus* were found on culture.

For two months the wound granulated slowly, and, as it filled up, the remains of the radius were gradually extruded, until at the end of four months a sequestrum composed of nearly the whole of the diaphysis of the radius, with the exception of small fragments continuous with each epiphysial line, was removed (*Fig.* 473). The granulations appeared healthy, with very little serous discharge.

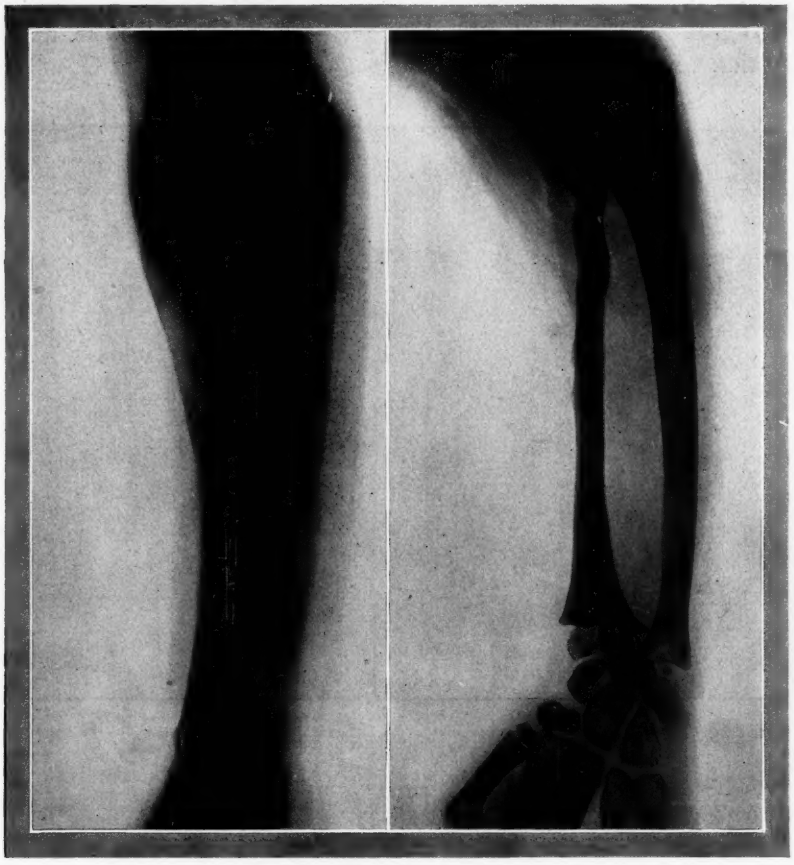


FIG. 477.—Development of a good anatomical radius, with survival of radial and ulnar epiphyses and all carpal bones (June 28, 1927). The irregularity of development of the lower radial epiphysial line has led to shortening and consequent 'radial deviation' deformity.

BONE-GRAFTING OPERATION.—With a possibility of limiting the deformity (radial deviation) and of quickening bone formation, bone-grafting was decided on, and, four months after the original operation, a graft 5 in. long from the right tibia was placed in a bed prepared between the extensor groups of muscles of the forearm and the flexor, wedged in between the lower and upper fragments of the radius, and fixed in position by deep catgut sutures (*Fig.* 474).

AFTER-HISTORY.—The wound healed by first intention, but after four

weeks a small sinus developed in the centre of the wound. No sequestrum was seen. Skiagrams showed absorption of the graft with new bone formation (*Fig.* 475, *a* and *b*). The sinus healed in two months, and gentle massage and re-educational exercises were commenced; but after some months of this the arm became painful, and on X-ray examination a fracture of the graft was found (*Fig.* 476, *a*). After three months' immobilization the fracture firmly united (*Fig.* 476, *b*), and massage and electrotherapy were recommenced, with application of radio-active mud and a vacuum electrode, in an attempt to free the scar, which had become adherent to the underlying bone. The child has attended regularly at the hospital since, and has worn a night splint to correct the radial deviation. (*Fig.* 477, taken in June, 1927, shows the formation of a new radius, and *Fig.* 478 the condition in October.)

FIG. 478.—Photographs showing the state of the forearms on Oct. 21, 1927.

The final report of the Electrotherapeutic Department three years after grafting, is: "*Elbow*: Extension full and flexion to 45°; pronation and supination both very weak. Some supination by biceps only. *Wrist*: Slight extension only, but wrist kept in dorsiflexed position owing to fibrosis of extensors and partial bony lock at wrist-joint. Good recovery in flexors and interossei, and very good grip of hand."

Although it will thus be seen that the child has fairly good power in the right hand, she has been, during convalescence, using her left, and it is doubtful at present which hand will be the leading member.

I wish to thank Mr. George E. Waugh for allowing me to perform the operations and for permission to publish an account of the progress and result of the case. The excellent series of radiograms was taken by Dr. Bertram Shires, to whom I am much indebted.

A SPINAL MENINGIOMA CONTAINING BONE.*

By LAMBERT ROGERS,
ASSISTANT IN THE SURGICAL UNIT, ROYAL INFIRMARY, CARDIFF.

This tumour, which I removed from a girl of 16, seems worthy of record because bone so rarely occurs in tumours of the spinal meninges.

History.—The patient was a schoolgirl who for two and a quarter years had suffered from pain in the back and right side below the costal margin. The onset of pain was followed by a slight limp, and gradually her legs became stiff. 'Tingling of the feet' developed, and was followed by a loss of sensation. For six months she had been unable to walk, and for three there had been incontinence of urine and fæces. Professor A. M. Kennedy, under whose care she was admitted, diagnosed spinal tumour involving the lower thoracic segments.

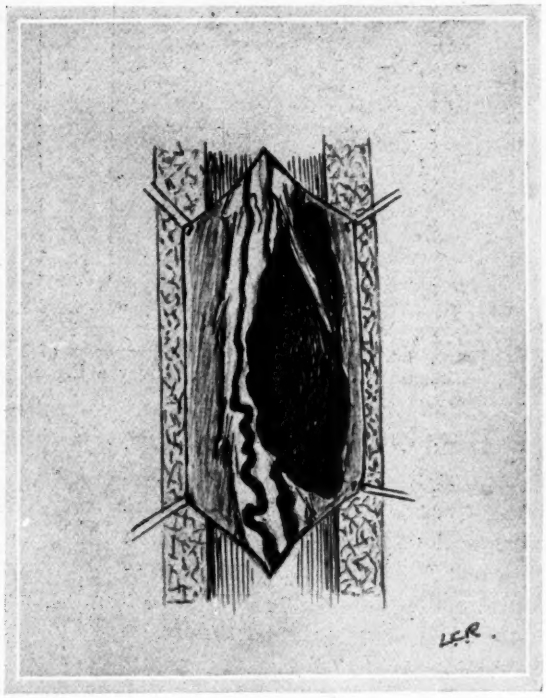


Fig. 479.—Drawing of the tumour as it appeared at operation.

Operation. — On opening the dura, laminectomy revealed an elongated, ovoid, maroon-coloured, lobulated tumour (*Fig.* 479) on the right posterior aspect of the cord, at the level of the 9th thoracic vertebral body. The tumour was intimately incorporated with the arachnoid and was slightly adherent to the dura, but easily separated from it. It measured almost 1½ in. in length, and flattened the cord to the shape of a strap.

The tumour was removed and recovery was remarkable, although the cord was so indented. At the end of three months the patient could walk unaided and had complete sphincter control. Eighteen months have passed since the operation and there is no evidence of recurrence.†

The tumour was found to consist of a very hard core surrounded by fleshy-looking lobulated masses similar to those usually seen in spinal endotheliomas. *Figs.* 480 and 481 show the microscopical appearances,

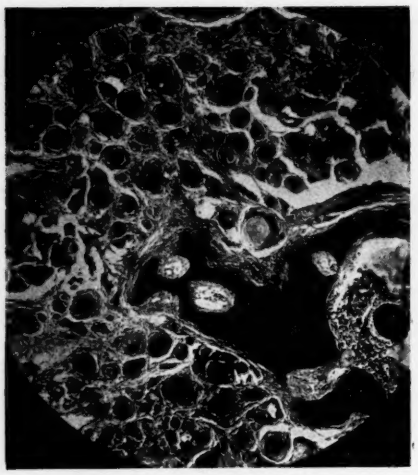
* From the Surgical Unit, the Welsh National School of Medicine, Royal Infirmary, Cardiff.

† This patient was shown at a Meeting of the South Wales and Monmouthshire Branch of the British Medical Association at Newport on Jan. 19, 1928.

numerous psammoma bodies lying in a fibrocellular matrix in which large, irregular masses of bone occur. *Fig.* 482 shows the X-ray appearances.

COMMENTARY.

The rarity of bone in spinal meningiomas is shown by the fact that Elsberg,[1] in his series of 100 spinal tumours published in 1925, makes no reference to bone in any of the 67 intrathecal and extramedullary tumours, 54 of which are given in detail, while Sir Percy Sargent[2] tells me that in his extensive experience he has never met with a bone-containing spinal meningeal tumour.



FIGS. 480, 481.—Microscopical appearances of bone-containing spinal meningioma removed at laminectomy. *Fig.* 481, taken under a higher power, shows the bone cells in the bone masses.

Dandy,[3] in one of his cases, describes a curious shell of bone lying over the dorsal part of the cord and extending from the 9th thoracic to the 1st lumbar vertebra. The bony mass was free within the dura except where it was attached loosely as the result of being tunnelled by a nerve-root. Five years after its removal a similar shell extending from the 2nd to the 5th lumbar vertebra was removed. Dandy regarded this 'pseudo-tumour', as he termed it, as of the nature of the pia-arachnoid plaques so frequently seen in explorations of the spinal cord. This bone shell bore no resemblance to the circumscribed meningioma recorded here.

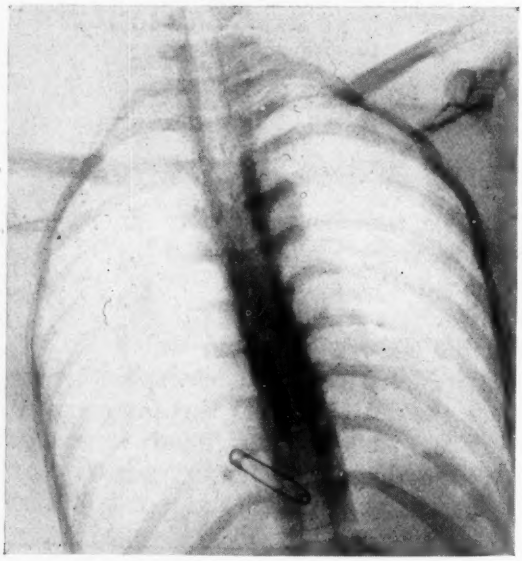


FIG. 482.—Radiogram of spine shortly after injecting lipiodol into the cisterna magna. The appearance was unchanged for twenty-four hours—complete spinal block.

Cushing,[4] believing that meningeal tumours, whether originating from the cells of the arachnoid villi at the points of emergence of the spinal nerves, or from those projecting into the venous sinuses of the cranial dura, are essentially similar, remarks that certain of the older and psammomatous growths may become fibrous and contain deposits of true bone, a condition known to occur within the skull.

The evidence of true bone formation in a spinal endothelioma presented by this case serves to emphasize the essential similarity of the cranial and spinal meningiomas.

REFERENCES.

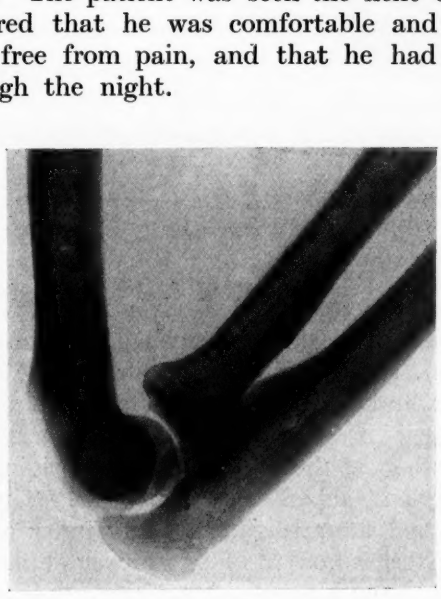
[1] Elsberg, C. A., *Tumours of the Spinal Cord*, London, H. K. Lewis, 1925.
[2] Sargent, Sir Percy, Personal communication.
[3] Dandy, W. E., *Ann. of Surg.*, 1925, lxxxv, .223.
[4] Cushing, H., *Intracranial Physiology and Surgery*, Oxford Medical Publications, 1925.

A CASE OF DISLOCATION OF THE ELBOW WITH SEPARATION OF THE INTERNAL EPICONDYLE AND DISPLACEMENT OF THE LATTER INTO THE JOINT CAVITY.

By H. B. WALKER,

SURGEON TO THE MINEHEAD AND WEST SOMERSET HOSPITAL.

R. S., a gardener, age 18, on Aug. 26, 1927, slipped on the wet pavement and fell on the point of his left elbow. The doctor who attended the injury described it as " an outward dislocation of the ulna with a forward displacement of the radius ". It was reduced only after the application of considerable force. The patient was seen the next day. He declared that he was comfortable and remarkably free from pain, and that he had been so through the night.



Figs. 483, 484.—Skiagrams taken after reduction but before operation.

On Examination.—There was a notable absence of swelling, considering the nature of the injury. Movements also were easy and caused little pain, though they were not pushed through any great range. The contours and shape of the joint were not greatly altered. On palpation a rough bony area was felt immediately beneath the skin over the internal condyle. Below this area the fleshy mass of the common flexors was not encountered. Had X-ray examination been impossible it is doubtful whether the above examination by itself would have rendered the imperative need for further treatment obvious to the observer. The X-ray film, however, showed a radial subluxation of both bones of the forearm. The lower part of the internal condyle was missing, whilst in the elbow-joint space, which was abnormally wide, and between the sigmoid notch of the ulna and the trochlear surface of the humerus, was a fragment of bone about the size and shape of the pisiform bone of the hand (*Figs.* 483, 484).

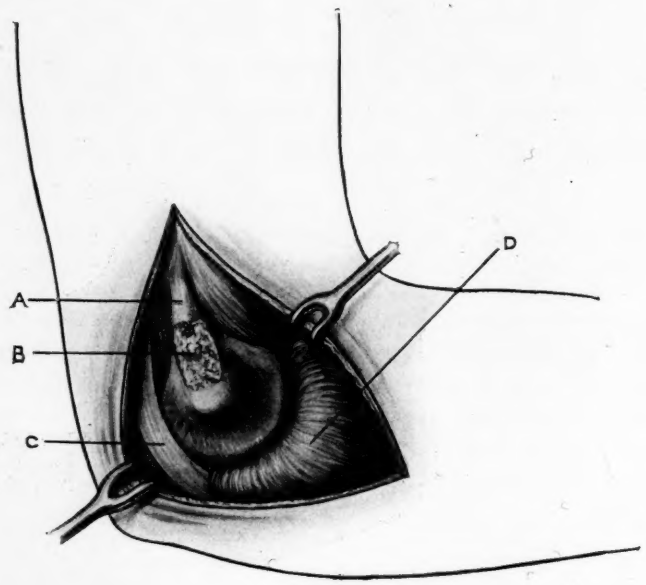
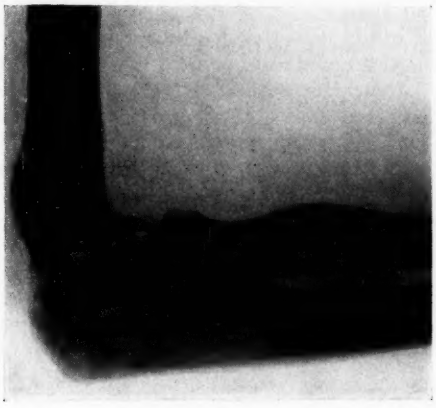


Fig. 485.—Diagram of condition found at operation. A, Intact upper part of internal condyle; B, Fractured area of internal condyle; C, Ulnar nerve (bruised and swollen); D, Common flexor origin turned into joint..

Operation.—On Sept. 1 the joint was opened through a curved incision on its inner side (*Fig.* 485). This exposed the fractured surface of the internal condyle immediately beneath the skin, and below this the inner face of the trochlear of the humerus. Below this and folded between it and the sigmoid notch of the ulna, so as to occupy the joint space between these two articular elements, was a muscular flap consisting of the common flexor origin, with the lower portion of the internal condyle attached to its extremity. The deep face of this flap consisted of the internal lateral ligaments and inner capsule of the elbow-joint. Beneath the posterior border of the muscular flap, the ulnar nerve, which showed considerable evidences of bruising, disappeared on its normal course. The muscular flap was hooked out of the joint cavity

and the separated fragment of the internal condyle was fixed in place by a suture of thick catgut passed through a hole bored in the humerus, and through the common flexor origin. Healing took place without event. Examination after the operation showed a definite ulnar paresis without any corresponding sensory changes. This was no doubt present from the time of the accident, but had remained unobserved through inadequate examination.

The patient insisted on resuming light work on Sept. 30, when the movements of the joint were good, though complete extension was not yet recovered. Weakness of the ulnar group of muscles still persisted on Oct. 25. Skiagrams taken on Oct. 25 show the internal condyle in correct position with the line of fracture still present (*Figs.* 486, 487).



FIGS. 486, 487.—Skiagrams taken seven weeks after operation.

COMMENT.—The mechanics of the production of the condition would seem to be simple. The external dislocation of the ulna carries outwards the separated internal condyle, which is caught between the bones and remains in the joint when reduction is effected.

The clinical points which struck the observer about this case were the absence of pain and effusion, and the ease with which voluntary movements were carried out within twenty-four hours of the injury. Possibly the interposition of the muscle flap between the articular surfaces was responsible for these effects. The ulnar nerve was obviously severely bruised at the time of the injury.

REVIEWS AND NOTICES OF BOOKS.

Diathermy: its Production and Uses in Medicine and Surgery. By ELKIN P. CUMBERBATCH, M.A., B.M. Oxon., D.M.R.E. Camb., M.R.C.P., Medical Officer in Charge of Electricity Department, St. Bartholomew's Hospital, etc. Second edition. Demy 8vo. Pp. 331 + xiii, illustrated. 1927. London: Wm. Heinemann (Medical Books) Ltd. 21s. net.

THE medical and surgical applications of diathermy have been considerably extended since the appearance of the first edition of Dr. Cumberbatch's book in 1921. There have been advances, not only in the technique and scope of the diathermic current, but also in the machines used for the generation of the current. In the first half of the book the author describes in detail the design of diathermy machines produced in this country, as well as those made by the leading manufacturers in America and on the Continent. Regarding the construction of diathermy machines, it is interesting to note that the principle of the thermionic valve, with which many are familiar in the form of a 'wireless valve', is being exploited in the latest type of diathermy generators, and that these valve machines have given considerable satisfaction. It appears probable that the majority of machines in the future will be constructed to incorporate this principle.

The latter half of the book is devoted to the therapeutic application of diathermy. 'Medical diathermy' the author defines as the 'heating through' of the body tissues to a temperature insufficient to destroy or impair their vitality. Of special interest is the author's technique and experience in applying diathermy to the treatment of inflammatory lesions of the female pelvis, and to infections of the urethra, prostate, and vesicles in the male. In gonorrhœal infections in the female he regards diathermy almost as a specific cure, but in the male as only an adjuvant to other treatment, although it is stated that, when the infection is limited to the anterior urethra, diathermy will almost certainly prevent extension of the disease to the prostate and vesicles.

Under the heading of 'Electro-thermic Methods of Surgery', which term the author prefers to 'surgical diathermy', are described three methods of using the diathermic current for the destruction of tissue: (1) Diathermic coagulation; (2) Electro-desiccation; and (3) Fulguration. Surgically the chief interest is centred in a modification of the last method, which is called the 'arc operation'. This procedure is not true 'diathermy', but is the utilization, for cutting purposes, of the spark or 'arc' which is formed when the active electrode is brought close to the tissues, but without actual contact. When the arc has once been formed, the active electrode can be inserted into the tissues and drawn forwards. The arc precedes the electrode and divides the tissues, sealing, at the same time, the divided surfaces. The margins of an area thus excised can be brought together by sutures, and the wound heals by 'first intention'. The method has been used for the removal of cancer of the breast, and it is claimed that skin recurrences are infrequent, owing to the immediate sealing of the tissue spaces as the operation proceeds.

In the treatment of malignant disease in certain sites diathermy is supplanting ordinary surgical methods, and particularly is this true of malignant disease of the mouth and tongue, where even extensive applications have been followed by comparatively little shock. The author quotes numerous authorities who are convinced that destruction of the primary growth in this way, with applications of X rays or radium to the cervical lymphatic glands, has given vastly better results than excision of the growth and the removal of the cervical glands.

Of the minor surgical uses of diathermy, attention may be drawn to the use of the current in coagulating small vessels, and thus avoiding the use of ligatures.

This can be done by lifting the artery forceps holding the divided end of a vessel, and touching the shaft of the forceps with the active electrode. The current then runs down to the blades and coagulates the tissue to which they have been applied.

In this country diathermy has not yet attained the position which it merits, in either its medical or surgical aspects. Extensive fields for the application of this current await investigation, such as the treatment of pneumonia, surgical shock, and eye diseases, to instance a few. It is to be hoped that Dr. Cumberbatch's excellent and well-illustrated book will stimulate further interest in the undoubted value of this method of treatment.

Plastic Surgery of the Head, Face, and Neck. By H. LYONS HUNT, M.D., L.R.C.S. (Edin.), Consulting Plastic Surgeon, Midtown Hospital, New York City, etc. Medium 8vo. Pp. 404 + xiv, with 342 engravings and 10 coloured plates. 1926. Philadelphia and New York: Lea & Febiger. $7.00.

THIS handy volume, profusely illustrated and containing a very full bibliography conveniently arranged at the end of each chapter, represents a useful addition to the rapidly increasing literature of this new special field of surgery. Throughout the work the author treats of cosmetic defects and deformities on the same footing as disfigurements produced by accident or disease. This is greatly to his credit, for we agree with him that in many instances the former are scarcely less tragic than the latter. There are twenty chapters, arranged where possible on uniform lines—surgical anatomy, study and analysis of the condition under treatment, principles involved in the treatment, and operative technique.

A very full historical review, pointing out the important part played by the world war in reawakening interest in the subject of plastic repair, ends by claiming equal rights to treatment for those who from birth have been victims of facial defects and for those who, having possessed regular features, refuse to tolerate disfigurement.

In an excellent chapter on 'General Considerations', types and lines of incision and methods of wound closure are dealt with in detail. The usefulness of so detailed a history-form as that which is here given, and which occupies no fewer than three pages, is not understood.

A soundly-supported condemnation of the use of hydrocarbon injections indicates that the author has had a wide experience of the devastating results of this 'easy' method of contour production. No space occupied by such a condemnation could be better used in a book of this type, for paraffinomata cases are the saddest, most unsatisfactory, and most difficult which come into the hands of the plastic surgeon.

Local anæsthesia is ably dealt with and fully described in a special chapter by Maxeiner.

While the use of moulds in Thiersch grafting in the mouth and eye regions is fully described in a later chapter, their use as advocated by Gillies, or the employment of the pressure bags suggested by Ferris Smith for the prevention of the accumulation of serum between the graft and its bed, appears to have been entirely overlooked in the chapter on 'Grafts and Transplants'.

Pleas for the avoidance of tension, for careful control of bleeding, for early removal of skin sutures, and for the post-operative use of the ice-bag are all of great importance; and the description of the author's experiments with different types of turtle bile, for bringing about absorption of the fibroblast-laden exudate from the edges of newly-closed wounds and so diminishing scar formation, makes interesting reading. This subject, which is intimately associated with the very important one of 'Keloid' (treated in the following chapter), is one in which there is room for much research.

The chapter on 'Scalp and Skull Defects' includes a description of 'forehead lifting'—called by the author 'frontal rhytidectomy'—for the elimination of wrinkles in this region. Eyelid defects are fully considered, but we find no mention of the use of mucous-membrane grafts in the treatment of entropion. Actual case-records of patients in whom eyelashes have been restored by any of the methods described

would have made a more convincing recommendation of the procedure than the bare text. The technique of purely cosmetic operations for the removal of eyelid wrinkles is given.

Ear defects, cosmetic operations on outstanding, over-large, and otherwise deformed ears, all forms of rhinoplasty, together with methods of treatment for hump-nose, saddle-back nose, rhinophyma, and alar deformities are all fully considered. Parotid fistula, cicatricial and bony trismus, fractures of the maxilla and mandible, and cosmetic skin-stretching operations for naso-labial skin redundancy fall within the scope of other chapters, and there is an excellent review of all recent work on hare-lip and cleft palate. We think the author gives sound advice when he says that those methods of hare-lip repair which involve complicated incisions are best avoided. In the chapter on 'Neck Defects', which is chiefly occupied with burn contractures, details of operations for the correction of laxity of submental skin and for double-chin deformity are included.

The book is rounded off by a chapter on 'Physiotherapy' which describes the treatment of thickened scars, keloids, telangiectases, and various forms of skin blemish by means other than surgery. The danger of producing skin atrophy, telangiectasis, or actual scarring by attempts to obtain permanent epilation by X rays is wisely stressed.

There is a certain impersonal element about the book as a whole which might have been obviated by the inclusion of more numerous records from the author's own practice. The publishers are to be congratulated on the excellence of the reproduction of the illustrations, and the only criticism we would offer is that coloured plates might have been employed to much better advantage in giving a clearer conception of the disfigurement produced by burns, paraffinomata, and the like, where ordinary photography fails so markedly, than in anatomical illustrations, reference to which in special books of anatomy is so easy.

Chirurgische Operationslehre. By Professor Dr. O. KLEINSCHMIDT, Wiesbaden. Royal 8vo. Pp. 1269, with 705 illustrations, many in colour. 1927. Berlin: Julius Springer. M. 57.

PROFESSOR KLEINSCHMIDT is a pupil of Professor Payr, of Leipzig, to whom he dedicates this work, and in it he represents the teaching of that surgeon. It is intended as a text-book for students and also for qualified practitioners, and in both these objects it is eminently successful. The very difficult problems of how much to include and how much to exclude have been wisely dealt with, so that the book will be of great value for reference and consultation. Its value as a work of reference is greatly enhanced by the well arranged bibliography at the end, which is placed under the headings of various operations and surgical diseases; but we notice that these references are almost entirely confined to German authors.

The introductory chapters on general surgical principles are clearly written, but are perhaps too abbreviated to be of much practical value. The chapter on vascular surgery is particularly well illustrated by coloured and semi-diagrammatic figures showing the anatomical relation of the principal arteries. Operations upon fractures are very shortly described, and the methods chiefly illustrated are those which involve the use of metal sutures. It is stated that the modern tendency in fracture operations is to impact the fragments and to avoid the use of any unnecessary foreign bodies. Amputation and excisions of joints are well dealt with, as are the various methods of arthroplasty, as might have been expected from an author who is the pupil of Payr.

In the sections dealing with the head and neck, the thorax, and the abdomen, all the classical operations are well described and illustrated. A certain number of rare and unusual operations are also included, e.g., Trendelenburg's operation for the removal of a pulmonary embolus, and Payr's operation for division of the falx cerebri. A long account is given of the various methods of prostatectomy, including the removal by the perineal route (Zuckerkandl) and Voelcker's method by the ischiorectal route.

The illustrations are very well drawn and excellently reproduced, but their distribution is very unequal. As a presentation of German operative methods we think this volume is of great value.

Appendicitis. By HUBERT ASHLEY ROYSTER, A.B., M.D., Surgeon to Rex Hospital; Surgeon-in-Chief to St. Agnes Hospital, Raleigh, N.C.; Former President, Southern Surgical Association. Royal 8vo. Pp. 370 + xiii, illustrated. 1927. London: D. Appleton & Co. 21s. net.

THE author points out that no treatise on the appendix has appeared within the past few years, and although many articles have been published in the medical journals on the subject during this period, yet there is need for their correlation and integration in view of enlarged experience. The present monograph attempts this, and is an endeavour to stabilize contemporary opinion upon the main features of interest in connection with appendicitis, more especially those with a practical bearing. With this object a careful review has been made of the more important papers published in the journals during the last five years, and the opinions of their writers upon the various aspects of appendicitis have been freely quoted. Although the author frequently expresses his own views, and declares his personal preference, founded on his individual experience, for certain methods of procedure, yet the work must be regarded mainly as a compendium of the opinions of others, and as such constitutes a most valuable book of reference.

Etiology is well considered, and some interesting instances are given of the relationship between appendicitis and trauma. The chapters on diagnosis and on the complications of appendicitis are particularly worthy of mention, and the special features of the disease when attacking young children are rightly and strongly emphasized. The X-ray as a diagnostic agent in chronic appendicitis is extensively discussed, and very diverse views are quoted; the author's conclusion that it is of limited value seems fully justified.

The Ochsner treatment receives full discussion, and it is pointed out that this method has been woefully misunderstood. Ochsner himself proposed it only for those border-line types which Maurice Richardson described as "too early for the late operation and too late for the early operation"; moreover, Ochsner never intended his treatment as a substitute for operation but as a preparation for it. The Murphy method of "getting in quick and getting out quicker" is also discussed. The author's conclusion is that "the question of when to operate in acute appendicitis can be decided only upon an individual basis. . . . Whether one should operate at once—the natural course, barring complications—or defer operation for any reason will depend upon the stage of the disease, the patient's general condition, the operator's choice of method suited to his skill. . . . The study of each patient's problem in the light of experience is the only satisfying recourse."

Cancer d'Estomac. By VICTOR PAUCHET and A. HIRCHBERG. Royal 8vo. Pp. 188, with 173 illustrations. 1928. Paris: Gaston Doin et Cie. Fr. 45.

THE clinical part of this book, for which Pauchet is responsible, is built up on the lines which other works by the same author have made familiar. That is, it is an atlas of operative treatment of cancer of the stomach, the letterpress being largely explanatory of these drawings. There is, however, an interesting chapter on prognosis, in which the author gives the results of 100 gastrectomies for carcinoma of the stomach. He had a mortality of 27 per cent. When there was free hydrochloric acid present it was only 10 per cent, but when there was anachlorhydria there was a death-rate of 44 per cent. Speaking of late results, he says that about one-third lived more than three years. These results he regards as poor, but thinks there would be many fewer cases if all ulcers of the stomach were excised. He lays great stress upon the use of local anæsthesia and of diathermy upon the operating table. Gastro-enterostomy prolongs life for about six months only.

The section by Hirchberg gives a good account of ulcer-cancer. The author

also draws attention to that form of cancer which is cauliflower-like in appearance, and is often situated upon the greater curvature. Although glands are usually enlarged they are often free from carcinoma. The prognosis is good. The micro-photographs in this section are not so good as the very beautiful drawings by Dupuy in the first part. The legends attached to *Figs.* 51 and 54 appear to be transposed.

La Vesicule biliaire. By M. CHIRAY, Professeur agrégé à la Faculté de Médecine de Paris; and I. PAVEL, Assistant universitaire à la Faculté de Médecine de Bucarest. With an account of Vesical Radiology by A. LOMON, Electro-Radiologiste des Hôpitaux de Paris. Royal 8vo. Pp. 568, with 130 illustrations and 4 coloured plates. 1927. Paris: Masson et Cie. Fr. 70.

WE have read this fine work with ease and pleasure. It was pleasurable because of the clear graceful language, the valuable illustrations, and the good general turn-out of the book. It was easy because the whole subject of the gall-bladder is presented with scholarly completeness and in neat sections.

The authors have enriched European medical literature with an encyclopædic monograph on the anatomy, physiology, and pathology of the gall-bladder. Their collaborator Lomon is responsible for an excellent chapter on the radiology of the organ and this will be read widely with profit. It is very modern and full of new diagrams and reproductions of skiagrams. It can be read separately from the other parts of the book.

The main body of the book is made up of Parts 3 and 4. Part 3 comprises a section on biliary colic, another on biliary insufficiency, and another on post-prandial diarrhœa: this last section contains much that is really new and surprising. Part 4 falls easily into two divisions, concerned respectively with calculous and non-calculous cholecystitis. The whole of this part is exceedingly useful for its conciseness, but much of it was long ago propounded by Moynihan. In keeping with the book's general quality of completeness there is a very long account of the different hypotheses relative to the formation of gall-stones.

Part 5 is devoted to therapeutic measures. The authors make much, both from general diagnostic and particular therapeutic aspects, of the method of biliary drainage which they wish to be called the method of Doyon-Meltzer-Lyon. Duodenal intubation and the introduction of magnesium sulphate near the sphincter of Oddi is described in great detail. Full instructions, with interpretations of the various results, are given in the matter of fractional biliary secretion analysis.

The authors have drawn freely from the world literature, and so the continuity of the book is broken every little while by huge blocks of bibliography. It is hard to find anything missing from this most learned and recondite book. Indeed, one of its smallest sections is yet the fullest and best account we have ever read of the 'strawberry' gall-bladder.

Traitement de l'Ulcus gastro-duodénal. By Professeur FIDEL FERNANDEZ-MARTINEZ, l'Hôpital Saint-Jean de Dieu, de Grenade (Espagne). Crown 8vo. Pp. 138. 1927. Paris: Masson et Cie. Fr. 12.

THE first part of this book gives a general survey of present-day views on the treatment of ulcer of the stomach. It deals with principles only. The second part describes the medical treatment recommended by the author. This comprises a very strict dietetic régime combined with alkalis. What is of interest to the surgeon is the duration of treatment. After a period lasting for several weeks the patient enters upon the fourth stage, in which he has a very restricted diet, has to rest for an hour after each meal, has to drink a litre of alkaline water every day, and is only allowed to walk a short distance a day. This stage lasts, according to Professor Fernandez-Martinez, about two years. This is the minimum time for treatment.

Putting aside the bad psychological effect of such a prolonged period of invalidism, how many men can afford to devote so much time to a cure when surgery can accomplish more in far less time and at a not very serious risk?

The author gives the following indications for operation: pain, intractable hæmorrhage, penetration of the ulcer into neighbouring structures, stenosis, biloculation, perigastritis, and fear of cancerous change. As to the particular method, he believes a large resection is indicated wherever possible. The next most useful method is that of Balfour. He thinks gastro-enterostomy should be reserved for cases in which there is a narrow pylorus or a duodenal ulcer. Subjects such as protein-shock therapy and vaccine treatment are described, and many formulæ (far too many formulæ) given for the relief of symptoms which may arise.

Handbuch der speziellen Chirurgie des Ohres und der oberen Luftwege. Edited by Professor F. BLUMENFEL, Wiesbaden; and Professor Dr. R. HOFFMANN, Dresden. Royal 8vo. Pp. 1080. Illustrated. 1927. Leipzig: J. A. Barth. M. 87; bound, M. 95.

THIS volume covers an extraordinarily wide field, including, as it does among its larger headings, plastic surgery, anæsthesia local and general, and X-ray and radium therapy.

It is perhaps unavoidable in a book of this description—a book of high aim—that there should be a certain inequality and that some articles should show a very definite superiority over others. One of the most admirable of these former is that contributed by Professor Bockenheimer and Dr. Stein, which deals very fully with plastic operations on the face. The illustrations to this article are excellent, and the markings of incisions for flaps are made remarkably clear.

The correction of nasal deformities is also gone into by Professor Joseph, with the most painstaking and instructive detail. Cosmetic operations on the face, and rhinoplasty, are also dealt with by Professor Bockenheimer and Dr. Stein at great, but most satisfactory, length, and are very well illustrated.

Professor Thost, on stenosis of the larynx, describes the treatment of this condition in a manner both lucid and instructive, while a discussion on the causation of stenosis is admirably full. There is, however, a long discussion on the subject of paraffin injection for the nose, in which nothing fresh would appear to have been said about this treatment, the use of which, in any case, has now been practically abandoned in this country.

The article on general anæsthesia is perhaps the least good in the volume—indeed, it may be described as definitely disappointing, gas-and-oxygen and intratracheal anæsthesia being only just mentioned. On the other hand, the local anæsthesia of the head, neck, and ears is well dealt with by Dr. Heyman and Dr. Voss, and the illustrations and landmarks for block anæsthesia are very clear and good.

The chapter by Dr. Pfeiffer on X-ray diagnosis in the upper air-passages and ear gives us a careful and most interesting précis of the positions in which the X rays should be taken for the various regions which are under examination, and the interpretations of the findings. The X-ray pictures here reproduced are extremely typical and quite excellent.

Treatment by means of X rays and radium is the subject of another chapter. This wide and most important subject, which is still in its infancy, and the conditions in which this form of therapy may be of use, are discussed at great length, as are details of dosage, methods of application of radium, and the form in which radium should be applied. Dr. Albanus is responsible for this chapter, which is most carefully dealt with and of great interest.

Much more might have been said on the septic conditions of the ear, nose, and throat. These subjects are, unfortunately, very hurriedly sketched in, and since they can in no possible wise be ignored, or lightly touched upon, in any volume which claims so comprehensive a grasp of the whole area of oto-rhino-laryngology as does this one, it must be said that this portion of the book is not up to the standard of the volume in general, which as one of reference in radiotherapy and plastic surgery is to be thoroughly recommended, and which fills a definite place in the literature of these subjects.

The work is very well illustrated, and there are copious references both at the foot of the page, and in greater detail at the end of each chapter. The indexing is adequate.

Die Chirurgie: A System of Surgery. Edited by Professors KIRSCHNER and NORDMANN. Imperial 8vo. Berlin: Urban & Schwarzenberg.

FASCICULUS 20, concluding Vol. IV. Pp. 188, with 103 illustrations in the text, some in colour, and 26 coloured plates. M. 19.

THIS section of the work, which is by Professor Jehn, of Mainz, and Professor Schubert, of Marienwerder, deals with the surgery of the lungs and the chest wall. It is remarkable for the great wealth and beauty of the coloured illustrations, which give most exact pictures of such conditions as gangrene, bronchiectasis, etc., and also of the many X-ray figures of the various lung diseases. There is a very clear and most valuable description of the various operations for the cure or relief of pulmonary tuberculosis, and a good critical summary of results in a large number of cases. In 1024 cases, 32 per cent were cured and 31 per cent died—12 per cent as the immediate result of the operation and 19 per cent at a later period.

The rare diseases of the lungs, new growths, and parasitic infections, are given a fair space, and the possibility of relieving emphysema by resection of the rib cartilages is described but not commended.

Professor Schubert's article on the affections of the chest wall deals chiefly with congenital malformations and the infections of the ribs by tubercle or other diseases.

FASCICULUS 21, Vol. II. Pp. 235, with 108 illustrations in the text, some in colour, and 16 coloured plates. M. 17.

THIS section is by Professor Müller, of Marburg, and deals with the surgery of the muscles, tendons, and fasciæ. The article opens with a short description of the anatomy and physiology, regeneration, degeneration, inflammatory and traumatic affections of the skeletal muscles. A great deal of this is of not much practical importance, but it gives a good foundation of normal physiology for later surgical deductions. In discussing ischæmic paralysis, we think that undue optimism is expressed about the operation for shortening the forearm bones, whilst the very simple and practical step of sliding downwards the common origin of the flexor muscles is not mentioned. The most valuable part of this section is that dealing with the various possibilities of tendon lengthening and tendon transplantation. We are glad to note that in regard to the inflammatory affections of the tendon sheaths, due reference is made to the work of Kanavel, because throughout most of this new text-book any but German authors are so seldom mentioned. The illustrations of the various operations for tendon transplantation are models of clearness and artistic beauty. We think that the value of this section of the work would be greatly increased if a more critical estimate were given of the practical results of the different operations described. It is generally recognized, for example, that tendon transplantation for musculospiral paralysis gives rapid and excellent results, whereas the many operations for paralysis of the foot muscles are most disappointing. The last part of this section describes the various uses to which free fascia transplantation may be put.

Frakturen und Luxationen: A Short Text-book for Students and Practitioners. By Dr. K. BAUER, Assistant Professor of Surgery, Göttingen. Royal 8vo. Pp. 236, with 237 illustrations. 1927. Berlin: Julius Springer. M. 15; bound, M. 16.80.

THIS is a very clearly written and well-illustrated description of fractures and dislocations. The introductory chapters, which deal with general principles of etiology, mechanism, diagnosis, and treatment are models of lucid description. The book includes the consideration of fractures of the skull and vertebral column; but these subjects involve such completely different principles from those of the injuries of the limb bones that we rather question the advisability of including them.

The ordinary methods of treatment by splints, plaster, and axial traction are well described, but we think the scope and methods of open operation are not sufficiently dealt with. Thus the advantages of bone-grafting operations in cases of

spinal fracture without cord lesion are not mentioned. Fractures of the neck of the femur are described without adequate reference to the possibilities of operative treatment. The illustrations throughout are beautifully clear and very well reproduced.

Technische Operationen in der Orthopädie. By Dr. Med. JULIUS FUCHS, Baden-Baden. Medium 8vo. Pp. 230, with 126 illustrations. 1927. Berlin: Julius Springer. M. 16.50; bound, M. 18.

THIS small monograph deals exclusively with the devising, making, and fitting of appliances for the correction of deformities or the compensation for paralysis. Details are given of how all the apparatus necessary can be made and fitted, including the working of the metal bands and joints. The materials chiefly used are moulded leather, steel rods, and elastic bands made of silk-covered rubber. With these materials all deformities and paralysis are treated, and a great wealth of detail in description and clear illustration is given, which makes the method clear. One is very favourably impressed with the exactness and scientific precision of Dr. Fuchs' methods, although the doubt arises whether the extensive close-fitting leather splints are suitable for permanent use. Again, is it necessary to use so much more difficult and expensive appliances for simple fractures when plaster bandages afford the same principle of fixation?

The most valuable sections, we think, are those which describe how contractures can be overcome or paralysed muscles replaced by the use of broad elastic bands which exercise constant traction that can be graduated both in amount and direction.

Le Traitement des Fractures du Col du Fémur par la Méthode du Professeur Delbet. By JACQUES LEVEUF and CH. GIRODE. Royal 8vo. Pp. 148, with 164 illustrations. 1927. Paris: Masson et Cie. Fr. 30.

THE arrangement and method of this small monograph make it an ideal of clear exposition. Professor Delbet had used his method before the war and has developed it greatly during and after that period. The method consists of two acts. By the first of these the fracture of the neck of the femur is reduced and fixed by an external apparatus under the guidance of the X rays; by the second the broken bone is transfixed by a screw or a graft guided into place by a special drill guide. The placing of the drill, screw, or graft is done through a small external incision without exposure of the fracture. No special fixation is used after the operation. After the eighth day the patient is allowed to move his leg; he stays six weeks in bed, and after three to four months is allowed to walk unsupported.

The great value of the book is the clear and honest account given of both the immediate and late results. One hundred and thirty-four cases are described, with 90 late results. The most startling thing is the mortality of over 9 per cent. These fatal cases were for the most part in elderly patients and after operations done for recent fractures. Of cervico-trochanteric fractures there were 24 operations with 4 deaths. It is admitted that as natural union by callus takes place in these, operation is not necessary, and it is no longer performed. Of 53 recent cases of fracture of the true neck of the femur, late results were known in 37, and of these, 8 were very good, 13 fair, and 16 bad. Of 29 cases of ununited fracture treated by a fibular graft the result was known in 22, and of these, 9 were good, 10 fair, and 3 bad. It is, however, to be noted that in many of those described as good or fair the X rays show a very poor position or bad union. In 15 cases a dead bone peg was used, and of these, 10 results were bad and only 1 was known to be good.

The book is of great value because it enables the reader to form so accurate an opinion of the method of treatment described. We must confess that the considerable mortality, the complexity of the apparatus, and the uncertainty of the results would make us hesitate to use the technique for operation or after-treatment which is advocated.

Etudes sur les Affections des Os et des Articulations. By ANDRÉ LÉRI, Professeur agrégé à la Faculté de Médecine de Paris. Medium 8vo. Pp. 460, with 128 illustrations. 1927. Paris: Masson et Cie. Fr. 58.

PROFESSOR LÉRI, who has already written a well-known work dealing with the affections of the vertebral column, now presents in this small volume a collection of essays on certain aspects of general bone and joint diseases. It is in no sense a formal description of the common diseases, but rather a special presentation of rare conditions or of special aspects of common diseases. Among the diseases of developmental or congenital origin the author describes generalized osseous dystrophy under the term 'familial pleonosteosis'. He also has sections dealing with hypochrondroplasia, cleidocranial dysostosis, facial hemiatrophy, oxycephaly, multiple exostoses, and anencephaly. Among the acquired diseases of bone he discusses rickets, osteomalacia, Paget's disease, von Recklinghausen's disease, and the syphilitic affections of bone. The section on articular diseases is confined almost entirely to rheumatoid arthritis, with special reference to the nodosities forming in the periarticular structures. The most valuable part of the work is the carefully written histories of individual cases illustrating the rarer conditions. The figures in the book do not do justice to the subject matter as they are very poorly reproduced.

A Treatise on Orthopædic Surgery. By ROYAL WHITMAN, M.D., M.R.C.S., F.A.C.S., Surgeon to the Hospital for the Ruptured and Crippled, etc., New York. Eighth edition. Royal 8vo. Pp. 1061 + xii, with 954 engravings. 1927. London: Henry Kimpton. 45s.

THE regularity with which new editions of this book appear bears testimony both to the popularity of the work and to the energy of its author. The matter added in the present edition consists largely of descriptions of new methods of treatment and additional illustrations bringing the subject well up to date. Indeed, if criticism were to be made, it might be said that some of the older sections could be pruned down to render the volume less bulky, although much of the old matter is of such great clinical interest and is so well written that it would be difficult to select passages for excision.

Among the many new ideas in treatment may be mentioned the Ghormley bivalve corset for treating spinal caries with exposure to sunlight, and various new operative procedures such as Smith-Peterson's and Verrall's methods for arthrodesing the sacro-iliac joint and Hibbs' method of extra-articular arthrodesis of the hips. Then there are excellent new diagrams of methods of arthroplasty of the knee (taken from Campbell's description of Putti's operation) and of the elbow (from MacAusland), and many smaller points such as the treatment of recent acute osteomyelitis by multiple drill-holes into the metaphysis (Clarence Starr). Indeed, the whole work shows how carefully the author follows up the recent literature of his subject and incorporates all that he feels to be valuable in his work. There are, of course, omissions—for example, the method of correcting ischæmic contracture by a muscle-slide operation is omitted, and the description of cystic and fibrocystic conditions in the bones is unduly brief considering the great interest taken in these conditions in recent years. On the whole perhaps the older methods of splinting, braces, and other mechanical devices receive an undue prominence, and more recent methods of physiotherapy by re-education, etc., get less than they deserve, but the work still retains its position as one of the best—if not the best—general orthopædic text-books in the English language for students, practitioners, and specialists.

Treatment by Manipulation: A Practical Handbook for the Practitioner and Student. By A. G. TIMBRELL FISHER, M.C., F.R.C.S. Second edition. Demy 8vo. Pp. 200 + xii, with 62 illustrations. London: H. K. Lewis & Co. Ltd. 9s. net.

THE author of this book has been gratified by the rapid circulation of the first edition and is therefore encouraged to enlarge and rewrite parts of it for this second edition. Special emphasis is laid upon the necessity to found treatment upon a

sound anatomical basis, and yet the author frequently allows himself to talk almost like the osteopaths, e.g., when he says (p. 180), " It is easy to see that a unilateral rotatory displacement of the innominate bone upon the sacrum of a fraction of a millimetre might cause acute pain and disability." After this alarming statement we are somewhat comforted to learn (p. 185) that by manipulation " relief is usually immediate, as once the displacement has been reduced there appears to be no tendency to recurrence." We think it would be much safer to attribute pain and its relief by manipulation to adhesions or inflammatory products and their dissolution, than to talk about mythical displacements which can neither be proved nor disproved. As we said in reviewing the first edition, the indications for methods of manipulation are well described.

La Transfusion du Sang de l'Animal à l'Homme. By RENÉ CRUCHET, A. RAGOT, and J. CAUSSIMON. Crown 8vo. Pp. 106 + x. 1928. Paris: Masson et Cie. Fr. 12.

IT is a little difficult to appreciate exactly what the authors of this monograph believe themselves to have achieved by their investigations. They have shown that the blood of one animal is toxic to one of another species, and fatal if sufficient be given. This was already known. They have also found that the foreign blood is tolerated better if it is injected very slowly into the recipient's veins. Their experience that the blood of one animal if given to another slowly and in small quantity is not necessarily fatal encouraged them to try the therapeutic effect of the blood of horses and sheep upon human beings. The reader's own blood runs cold as he reads the accounts of how severely the patients suffered from the expected symptoms, and how narrowly they escaped death, and he is not consoled by the preliminary statement that " Le mouton choisi était superbe ". No evidence is forthcoming that any improvement that the patients may have experienced was due to the treatment. The whole treatise is in any case irrelevant to the larger problem of the therapeutic uses of blood transfusion. It is quite evident that the intravenous injection of animal's blood is merely one way, not without dangers, of producing protein shock. The quantity that can be given without killing the patient is very small, and can have no therapeutic value *qua* blood. All the corpuscles contained in it are, of course, immediately destroyed.

The authors nevertheless suggest in conclusion that the transfusion of blood from animals to man may soon fulfil a 'rôle énorme' in the treatment of disease. They have visions of stables filled with horses and of 'moutons superbes' immunized against every form of bacterial infection, including filter-passing organisms, with rivers of blood pouring from them into the veins of suffering humanity. We fear that they are, to say the least of it, *sanguine*.

Die Bluttransfusion. By Dr. E. KUBÁNYI, University of Budapest. With a Foreword by Prof. Dr. L. v. BAKAY, Budapest. Royal 8vo. Pp. 127, illustrated. 1928. Berlin and Vienna: Urban and Schwarzenberg. M. 6.

THIS excellent monograph, written in Budapest, follows the usual lines in giving a comprehensive account of the subject. It aims at gathering together scattered knowledge in reasonable compass, rather than at putting forward any original matter. The author writes partly from his own experience, and himself favours a technique which uses citrated blood. He devotes considerable space, however, to a description of various syringe methods of transferring untreated blood from donor to recipient. A very large number of authorities are quoted, but the author does not appear to have noted the work of Storer and Almroth Wright upon immuno-transfusion with defibrinated blood. He states, indeed, that the use of defibrinated blood is to be wholly condemned, a conclusion quite at variance with that of the authorities mentioned.

The book is in most respects a reliable guide to the subject, and it is well illustrated.

Die Lagerung verletzter und erkrankter Gliedmassen Leerschienen und verbandlose Wundbehandlung. By Professor D. Heinrich Braun, Zwickau. Royal 8vo. Pp. 118, with 101 illustrations. 1928. Leipzig: J. A. Barth. M. 12.

Professor Braun's wire splints became widely known during the war, and their utility was so generally recognized that they have now come to be used for the treatment of injuries and inflammations of the limbs in civil hospitals. In addition to the fixation of the limb, the wire splints enable the wound to be exposed without bandages or dressings, and the author extends this idea to other parts, e.g., the head, chest, or back, so that, by suitable wire cages, affected parts may be protected and yet exposed to the action of the light and air. In addition to this, the bulk of the book is occupied in describing the technique of fracture treatment by means of ice-tongs traction and the support of the various types of wire cradle splints. The illustrations, both photographs and diagrams, are models of clearness.

Lehrbuch der Röntgendiagnostik mit besonderen Berücksichtigung der Chirurgie. By H. R. Schinz, with the assistance of W. Baensch and G. Friedl. Imperial 8vo. Pp. 1131 + xviii, with 1722 illustrations, some in colour, and 5 photographic plates. 1928. Leipzig: Georg Thieme. M. 90; bound, M. 98.

It is seldom that the reviewer has a volume before him on which he can bestow so much unstinted praise as is the case with this text-book of radiographic diagnosis. It is not merely that the whole range of the subject is so carefully and systematically dealt with, but such an admirable balance is kept between the technical exposition of X rays on the one side and scientific medicine and surgery on the other.

The book is much more than an atlas of X-ray pictures, although the excellent figures make it in this respect a most valuable book of reference. Each subject is discussed primarily from the point of view of clinical diagnosis; then the special methods of X-ray examination are described and the results given. Subjects such as fractures in which diagnosis does not present any special difficulty are dealt with comparatively shortly, special attention being given to matters of doubt, as, for example, the formation and absorption of callus. Disorders of bone growth are fully discussed, and in this and other parts describing bone changes there are many valuable microscopical sections of diseased tissues to elucidate the X-ray appearances. The chapter describing changes in the skull, accessory nasal sinuses, and the ear are of very great value, giving as they do so many pictures of both rare and typical diseases and the results of ventriculography. The same may be said about the chapters on the radiological examination of the chest and abdomen, and it is difficult to do justice to these in the short space available for review.

The book should certainly find a place in every medical library, and in the interests of English readers it is to be hoped that the publishers will be able to arrange for an English translation.

The Methods of Clinical Diagnosis. By A. G. Gibson, M.D., F.R.C.P., Hon. Physician to the Radcliffe Infirmary, Oxford, etc.; and W. T. Collier, M.D., M.R.C.P., Hon. Assistant Physician to the Radcliffe Infirmary. Crown 8vo. Pp. 398 + xviii, with 136 illustrations and 3 coloured plates. 1927. London: Edward Arnold & Co. 12s. 6d. net.

This book is intended primarily for students during their ward work. It gives a very clear and careful account of the various methods of clinical examination, the greatest stress being laid on the simplest methods. The most important laboratory tests are described as well, so that it also affords a guide to clinical pathology. As the book is primarily intended for medical rather than surgical work, perhaps we ought not to comment upon it in detail, but we cannot refrain from expressing surprise that radiography is so very briefly discussed that it has not been necessary to insert a single radiogram to illustrate the diagnosis of diseases of the gastro-intestinal tract, chest, or skeleton.

Minor Surgery and Bandaging. By GWYNNE WILLIAMS, M.S., F.R.C.S., Surgeon, University College Hospital. Nineteenth edition. Crown 8vo. Pp. 439 + viii, with 247 illustrations. 1927. London: J. & A. Churchill. 10s. 6d. net.

THIS is the descendant of the book bearing the same title written by Christopher Heath in 1861. It still remains one of the best of its kind, and the present author is to be congratulated on the way in which he has included all that is important without making the book too bulky. Fractures are dealt with at some length, but we think that methods of traction by transfixion pins or calipers might have been given more fully. Some of the apparatus illustrated is obsolete, e.g., Petit's tourniquet or the pattern of Thomas's splint shown in *Fig.* 224. There is a very useful chapter on anæsthesia and local analgesia, but here again, in a book dealing with essentials, we think that the Vernon-Harcourt apparatus might have been omitted.

Travaux de la Clinique chirurgicale et du Centre anticancéreux de la Salpêtrière. Edited by A. GOSSET, Professeur de Clinique chirurgicale à la Facultè de Médecine de Paris; Chirurgien de la Salpêtrière. Imperial 8vo. Pp. 275, with 134 illustrations. 1927. Paris: Masson et Cie. Fr. 65.

THE first volume of the work of the Surgical Clinic of the Salpêtrière was published by Gosset a year ago and was favourably commented on in the pages of this journal. The second volume, which has recently been published, appears to be as good as the first. The following list of contents speaks for the wide interest taken in surgical matters: (1) Organization of the Surgical Clinic of the Salpêtrière, A. Gosset; (2) Surgical Treatment of Carcinoma of the Breast, A. Gosset; (3) Excision in two stages of Tumours of the Ascending Colon, A. Gosset and J. Charrier; (4) Degastro-enterostomy, Marcel Thalheimer; (5) Strawberry Gall-bladder, A. Gosset, Ivan Bertrand, and Georges Loewy; (6) An Account of 24,000 Anæsthesias, Dr. Maurice Boureau; (7) The Results of 75 Cases of Carcinoma of the Uterus treated by Radium and Surgery, Robert Monod; (8) Anatomical and Bacteriological Researches on Cancer of Plants, J. Magrou; (9) Surgical Treatment of Cancer of Glands of the Neck, Raymond Bernard; (10) Chronic Hæmorrhagic and Purulent Inflammation of the Rectum, Dr. Roger Savignac. All these are good articles and worthy of the attention of surgeons.

It is interesting to survey the ideals of Professor Gosset concerning the organization and constitution of a surgical clinic. In his article he gives an account of its inception and early work under Terrier and subsequent chiefs, and its present recent constitution after re-modelling. Gosset's ideal, which he has now been able to realize, is a number of laboratories grouped round the hospital and operating theatres so as to allow easy access and intercommunication to researchers and clinicians. The various laboratories which are now so grouped round the central service are: laboratory of biology, of chemistry, of bacteriology and hæmatology, of experimental surgery, of photography, a library, a service of radiology, a service of radiotherapy, and a laboratory of botany.

Readers of this review may possibly feel disposed to pay a visit to this Clinic, and, if they do, will not fail to be interested and stimulated.

The Surgical Treatment of Malignant Diseases. By Sir HOLBURT J. WARING, M.S., M.B., B.Sc., F.R.C.S., Surgeon to and Joint Lecturer in Surgery at St. Bartholomew's Hospital and Medical College, etc. Royal 8vo. Pp. 67 + xx, with 277 illustrations and 19 coloured plates. 1928. London: Humphrey Milford. 50s. net.

THIS volume comprises the Bradshaw Lecture of 1921 and a large amount of material collected for the purpose of the lecture; the whole subject has been brought up to date by the inclusion of Gye and Barnard's work and the Blair Bell treatment. It reviews the general pathology of the various types of malignant disease in different parts of the body, and outlines the operative and other methods by which they usually can be treated; statistics dealing with the etiology and

results of some operations are included. It comprises a careful and thorough piece of work, which from its nature must have occupied a large amount of time. It is well and suitably illustrated, and the coloured plates are unusually good.

A Text-book of Urology. By OSWALD S. LOWSLEY, A.B., M.D., F.A.C.S., Director of the Department of Urology of the New York Hospital, etc.; and T. J. KIRWIN, Ph.C., B.S., M.A., M.D., Chief of Clinic of the Department of Urology of the New York Hospital. Medium 8vo. Pp. 699 + xviii, with 233 engravings and 13 plates. 1926. Philadelphia and New York: Lea & Febiger. $10.

THIS is a book which will be found to contain an immense amount of information and a very extensive bibliography. It is divided into three parts: the first deals with the history of urological surgery; the second contains chapters on history-taking, physical examination, cystoscopy, and X-ray diagnosis; the third and largest part is concerned with the diseases of the genito-urinary organs. In this third section each organ is discussed very fully; its development, anatomy and physiology, and anomalies are described, and there follows an account of its diseases, with their pathology, diagnosis, and treatment.

The authors state in their preface that their purpose has been to "construct a complete text-book in one volume". Whilst trying to give them full credit for the immense labour involved in collecting so much information, we incline to think that they have occasionally neglected to read, mark, and inwardly digest this vast pabulum. Thus, if we consider the chapter on the prostate, we shall find a detailed account of the pathology of the disease known as adenomatous hypertrophy, but nothing about the effects this enlargement produces on the bladder, the ureters, and the kidneys; twenty-nine pages are devoted to the treatment of prostatic disease, but only fourteen lines to the indications for such treatment; so obsessed are the authors with the importance of pathology and treatment that they have omitted to give any account of the symptoms produced by adenomatous hypertrophy.

We have been unable to find any discussion of the value of lithotrity in the chapter on the bladder; this seems to us a surprising omission; can it be that Bigelow has been forgotten in America? On the whole we think not, for we have found two figures (154, 155) which do actually illustrate this operation, though there is no text to correspond to these pictures. The whole subject of stone and foreign bodies in the bladder is dealt with in two pages, whilst twenty pages are devoted to the subject of Cowper's glands.

A very full account is given of testicle transplantation. After reading the authors' description of the various operations which have been devised, and the evidence they accept as proof of the efficiency of such procedures, we find ourselves in complete agreement with their preliminary statement that "serious students and investigators have refused to consider the subject as coming within the scope of a treatise on urological surgery".

There are some very good illustrations, but we fail to see why a plate showing the perineal operation of prostatectomy should be inserted in the chapter devoted to the diseases of the ureter.

On the whole, we cannot commend this book to our readers, for it suffers from two grievous defects: it shows a notable lack of proportion, and gives the impression of having been written in a hurry.

Organisation und ordnungsgemässer Betrieb des Operationssaals. By Professor Max KAPPIS, Hanover. Demy 8vo. Pp. 119, with 28 illustrations. 1927. Leipzig: Georg Thieme. M. 4.80; bound, M. 6.

THIS is a small but very valuable practical guide to the organization and arrangement of the operating theatre and the technique of major operations. It discusses the different problems presented by the large and the small hospital or nursing home, and it describes the apparatus and technique in general use in Germany, which do not differ in any important particular from those which are found in this country.

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